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THE CALCIUM AND PHOSPHORUS CONTENT OF MILK FROM AUSTRALIAN WOMEN.

By BEVERLEY V. RITCHIE, B.Sc.,[1]
Australian Institute of Anatomy, Canberra.

THE calcium and phosphorus content of maternal milk has been estimated during the past half-century by many overseas workers, but no figures for Australia were available when required for another piece of research. Accordingly this investigation was planned and undertaken.

Previous Work.

Tables for calcium and phosphorus values drawn up by Widdows *et alii*[(1)] and Holt[(2)] have been compounded into Tables I, II and III, divided according to the secretion from (*a*) the colostral period, (*b*) the end of the first to the beginning of the tenth month, and (*c*) the tenth month onwards.

These tables illustrate the necessity for analyses of a large number of samples if any comparison of different workers' results is to be drawn.

This Investigation.

The Source of Material.

All samples of milk were obtained from women who were in regular attendance at the three mothercraft centres of the Canberra Mothercraft Society. It has already been observed[(3)] that the clientele of these centres is representative of an average Australian population, except that the frankly under-privileged are not found there.

Method of Collection of Milk.

The infants of these women were subjects of another investigation, and each mother was asked to bring a sample of milk with her when the infant was brought for monthly examination by the doctor. In many instances samples were collected at more frequent intervals, especially when a sample was found to be poor in calcium.

The women were provided with a sterilized wide-mouthed bottle of 30 cubic centimetres capacity fitted with a glass stopper, and were asked to express milk from the "middle" of the 6 a.m., 10 a.m., and 2 p.m. feeds on the day on which the sample was collected. With regard to the words "middle of the feed", it was explained that the mother was to allow the infant to drink for five minutes from one breast and then express about five cubic centimetres of milk and allow the infant to drink for a further five minutes; the procedure was repeated on the other breast. This method was adopted to enable the women to bring the milk with them when making a regular visit to the mothercraft centre.

The analysis of the milk was commenced the same afternoon.

Technique of Calcium and Phosphorus Determination.

The analysis was carried out on the ash from about 15 millilitres of the milk. This allowed for a duplicate estimation from the 30 cubic centimetres collected. The calcium was determined according to the Association of Official Agricultural Chemists' micro-method[(4)] with the following modifications:

The milk was evaporated to dryness on the water bath before being ignited in the muffle furnace, and the ash was then taken up in 15 millilitres of distilled water and 15 millilitres of hydrochloric acid. After having been heated on the water bath for twenty minutes the solution was made alkaline to phenolphthalein with saturated sodium hydroxide solution and then slightly acid with hydrochloric acid, and was then made up to 25 millilitres. Ten millilitres aliquot were taken for a determination.

The phosphorus was determined on five millilitres aliquot by means of Brigg's[(5)] method. Solutions were compared in a photoelectric colorimeter.

[1] Financed by a grant from the National Health and Medical Research Council and working under the direction of its Nutrition Committee.

TABLE I.
Showing the Mean Calcium and Phosphorus Values for Colostrum Obtained by Overseas Workers.

Author.	Age of Infant. (Days.)	Nature of Sample.	Calcium. (Milligrammes per 100 Millilitres.)	Phosphorus. (Milligrammes per 100 Millilitres.)
Widdows *et alii*[1]	0–14	101 subjects	33·3	15·9 (14 for 4–14 days)
Telfer[2]	5–12	5 subjects	28·4	20·9
Holt[1]	3–12	5 subjects (composite)	31·9	17·7
Soldner[1]	5–9	6–8 subjects (composite)	27·4	13·7
De Lange[1] ..	4–10	23 subjects (composite)	29·3	25·3
Schloss[1]	11–13	1 subject	23·9	16·6
Bahrdt and Edelstein[1]		4 subjects (composite)	34·0	—
Sikes	1–14	45 subjects	30·1	13·0

[1] Taken from Holt.[3]

TABLE II.
Showing the Mean Calcium and Phosphorus Values for Human Milk from End of First to Beginning of Tenth Month. Results of Overseas Investigations.

Author.	Age in Months.	Nature of Sample.	Calcium. (Milligrammes per 100 Millilitres.)	Phosphorus. (Milligrammes per 100 Millilitres.)
Holt[3]	1–9	Mean, 17 subjects	33·7	14·9
De Buys and Von Meysenbug[3]	1–7	Mean, 58 subjects	29·6	—
Schloss[1]	2–4·5	Mean, 4 subjects	31·8	18·4
Widdows *et alii*[1]	5–10	Mean, 17 subjects	35·6	17·9
Telfer[2]	2–10	Mean, 14 subjects	33·4	17·6[2]
Telfer[2]	2–10	Mean, 23 subjects[3]	33·2	—

[1] Taken from Holt.[3] [2] Rhachitic [3] Two to eight months.

TABLE III.
Showing Average Calcium and Phosphorus Value of Human Milk Collected during Late Lactation. Results of Overseas Workers.

Author.	Age in Months.	Nature of Sample.	Calcium. (Milligrammes per 100 Millilitres.)	Phosphorus. (Milligrammes per 100 Millilitres.)
De Buys and Von Meysenbug[3] ..	8–12	Mean, 12 subjects	26·2	—
Schloss	11–14	Mean, 3 subjects	20·7	16·8
Telfer[2]	12–13	Mean, 7 subjects	33·8	—
	10–20	Mean, 4 subjects	31·4	16·5
		1 subject[1]	34·3	—
Holt[3]	10–20	Mean, 10 subjects	27·9	13·3

[1] Rhachitic.

The Results.

Statistics.

During the past fifteen months 467 determinations were made on milk from 165 different women. In some cases a single analysis only was made of the milk from a woman. This was due to departure from the district or to sudden weaning *et cetera*. When the mother continued to attend the centre and was feeding her baby, in most cases a series of analyses was made at regular intervals. The number of women of whose milk 1, 2, 3 . . . 10 analyses were made is shown in Table IV.

The Calcium and Phosphorus Values.

From the series covered in this investigation the calcium content of milk from Australian women was found to vary from 17 milligrammes to 42 milligrammes per 100 millilitres. The phosphorus content varied from 7 milligrammes to 21 milligrammes per 100 millilitres.

For reasons discussed in the next section, no attempt was made at this stage to strike a mean value for either the calcium or phosphorus.

TABLE IV.
Distribution of Estimations.

Estimations Made.	Number of Women.
1	46
2	42
3	23
4	20
5	13
6	8
7	3
8	4
9	1
10	1

Calcium and Phosphorus Content in Relation to Period in Lactation.

Most authors[1][2][3][9][10][12] agree that as lactation progresses the calcium content of milk falls. I have tabulated my results according to the infant's age. These figures are given in Table V.

TABLE V.
The Calcium and Phosphorus Content of Milk According to the Age of the Infant.

Age of Infant in Lunar Months.	Number of Subjects.	Calcium. (Milligrammes per 100 Millilitres.)	Phosphorus. (Milligrammes per 100 Millilitres.)	Calcium:Phosphorus Ratio.
1	33	28·2	15·9	1·8
2	67	29·2	15·0	1·9
3	60	29·6	14·1	2·1
4	66	29·3	13·7	2·1
5	59	27·7	13·9	2·0
6	45	27·1	13·0	2·1
7	36	26·7	12·8	2·1
8	28	26·2	13·5	1·9
9	20	24·8	13·4	1·8
10	15	23·3	12·2	1·9
11	4	27·5	12·6	2·2
12	1	31·6	16·7	1·9

In this series the mean calcium value rose from the first month to a plateau through the second, third and fourth months and then progressively declined as lactation advanced. This is well shown in Figure I. In the five cases investigated after the tenth lunar month a substantial rise in calcium value occurred.

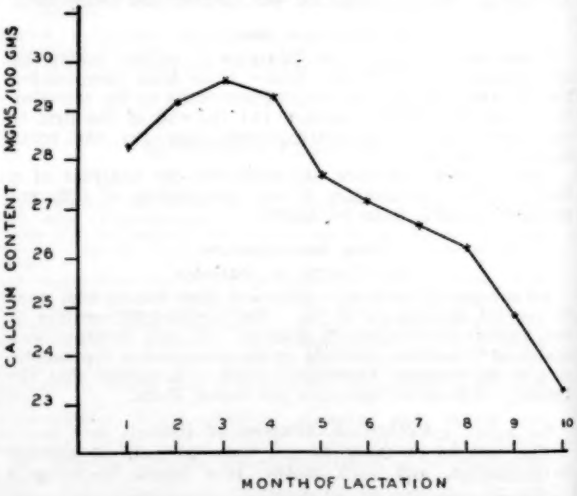

FIGURE I.
Mean calcium values at different months of lactation; for number of cases for different months of lactation, see Table V.

The phosphorus value appeared to fall steadily from the first month until the eighth, when it rose, and fell again after the ninth. Schloss,[12] Widdows *et alii*[13] and Telfer[32] made a similar observation.

Although a variability in the calcium:phosphorus ratio has been remarked by Camerer and Soldner,[34] by Schabad,[33], by Schloss,[12] by Burhans and Smith,[10] by Widdows *et alii*[13] and by Nims *et alii*,[9] I found that the average ratio was fairly constant over the whole period of lactation. The number of cases investigated in the eleventh month is too small for any inference to be drawn.

Calcium Content of Milk in Respect to the Season of the Year.

The absorption of calcium from the alimentary canal of an infant or an adult is influenced by the amount of vitamin *D* in the system.[18] As an individual may obtain vitamin *D* by the action of sunlight upon the skin, I decided to investigate the effect of season upon the calcium content of the milk.

Any consideration of the effects of the time of year will be influenced by the period of lactation, for, as I have already shown, the calcium content of maternal milk falls as lactation advances beyond the fourth month. Reference to Figure I will show that the calcium content forms a plateau from the second to the fourth month inclusive, and I decided to use the value for these months in the investigation of the influence of season.

The results for each stage of lactation and the average calcium figures for the different months of the year are shown in Table VI, from which it may be seen that the calcium content of maternal milk reached a peak in October and declined to a trough in May.

TABLE VI.

Average Calcium Value during Second, Third and Fourth Months of Lactation During Different Months of the Year.

Month of Year.	Number of Cases.	Average Calcium Value. (Milligrammes per 100 Millilitres.)	Standard Error.
1940—			
October ..	6	32·3	1·0
November ..	15	31·7	0·8
December ..	9	27·5	0·8
1941—			
January ..	24	29·3	6·8
February ..	23	30·1	0·8
March ..	18	29·1	0·8
April ..	14	30·0	1·4
May	15	28·3	1·0
June	12	29·2	1·3
July	20	29·3	1·0
August ..	14	28·5	1·1
September ..	20	29·3	0·9
October ..	9	29·2	0·9

The differences between the various months are not significant, but they show the possibility of climate as a factor influencing calcium content. This could be confirmed only by a larger series of cases.

Burhans and Smith[10] in 1923 suspected that there might be a possible decrease in the calcium content of human milk in the latter part of the winter and early spring, the time when rickets and tetany are most prevalent, and they quoted a small number of cases.

The Influence of Parity on the Calcium Content of Maternal Milk.

The distribution of the mothers with respect to the number of their children at the occasion of the investigation is shown in Table VII. The numbers after the fourth

TABLE VII.

Position of Child.	Number of Mothers.
First	98
Second	35
Third	14
Fourth	4
Fifth	3
Sixth	1

child are too small for these cases to be considered. The mean calcium value of the maternal milk for each month of age of the infants for the first, second and third babies is given in Table VIII.

The calcium values seem to be higher during the second lactation than during either the first or the third. The greatest single difference in an appreciable number of cases is at the second month between the second and third infants. This difference is equal to 4·0 milligrammes *per centum* and has a standard error of ± 1·5; it is accordingly not significant.

The greatest difference in phosphorus values occurs also at the second month in lactation between the second and third infants. The difference is equal to 2·6 milligrammes *per centum*, with a standard error of ± 0·64; it is therefore statistically significant.

On the numbers in this series no statement can be made concerning the influence of parity on the calcium and phosphorus content of maternal milk.

The Influence of the Early Onset of Menstruation after Parturition on the Calcium and Phosphorus Content of Maternal Milk.

From time to time during the past year I gathered the impression that often mothers whose milk had a low calcium value were also women whose menses had returned very soon after the birth of the baby. This inspired a closer examination of this aspect of the problem.

In Table IX the mean calcium and phosphorus content of the milk of 86 women is grouped into classes according

TABLE VIII.

Calcium Values of Maternal Milk During Lunar Months According to Number of Children.

Month of Lactation.	First Child (98 Women.)			Second Child (35 Women.)			Third Child (14 Women.)		
	Number of Samples.	Mean Calcium Value. (Milligrammes per 100 Millilitres.)	Mean Phosphorus Value. (Milligrammes per 100 Millilitres.)	Number of Samples.	Mean Calcium Value. (Milligrammes per 100 Millilitres.)	Mean Phosphorus Value. (Milligrammes per 100 Millilitres.)	Number of Samples.	Mean Calcium Value. (Milligrammes per 100 Millilitres.)	Mean Phosphorus Value. (Milligrammes per 100 Millilitres.)
1	14	27·9	16·1 (13)	7	27·6	15·6	3	28·0	15·7
2	38	29·2	15·4	12	30·7	15·2 (13)	6	26·7	12·6
3	40	29·6	14·2 (43)	9	30·8	15·1 (10)	6	28·1	13·6
4	45	29·2	13·8 (47)	10	30·7	14·5 (11)	4	26·1	12·8
5	37	27·4	14·1 (38)	9	29·9	14·3	4	25·1	12·4 (5)
6	28	26·4	12·6	9	27·8	14·2	4	26·6	11·9
7	24	26·3	12·9 (23)	8	27·3	12·4	2	24·9	12·3
8	17	25·6	13·0	6	27·3	14·1	2	23·4	11·7
9	13	24·5	13·5 (12)	4	26·8	12·6	1	24·1	—
10	10	22·1	12·8 (9)	4	24·6	12·5	—	—	—
11	3	28·4	12·0	1	24·6	14·4	—	—	—
12	1	31·6	16·7	—	—	—	—	—	—

TABLE IX.
The Influence of Menstruation during Lactation on the Calcium and Phosphorus Content of Maternal Milk.

Age of Infant in Lunar Months.	Mean Calcium Value from Table V.	Mean Phosphorus Value from Table V.	Age (Months) of Infant at which Mother's Menstruation Commenced.											
			0 to 1 (13 Subjects).			1 to 4 (22 Subjects).			4 to 6 (28 Subjects).			6+ (28 Subjects).		
			Number of Samples.	Mean Calcium Content.[1]	Mean Phosphorus Content.[1]	Number of Samples.	Mean Calcium Content.[1]	Mean Phosphorus Content.[1]	Number of Samples.	Mean Calcium Content.[1]	Mean Phosphorus Content.[1]	Number of Samples.	Mean Calcium Content.[1]	Mean Phosphorus Content.[1]
1	28·2	15·9	2	24·5	14·5	2	28·5	15·3	4	25·0	15·9	4	31·2	16·1
2	29·2	15·0	10	27·8	14·8	9	28·6	14·4	7	28·2	13·3	14	30·0	14·7
3	29·6	14·1	8	27·9	14·3	10	29·6	15·1	11	28·7	14·6	14	31·6	14·2
4	29·3	13·7	8	27·2	13·9	11	30·6	13·9	9	28·1	12·9	14	30·0	14·5
5	27·7	13·9	9	25·7	14·2	7	28·3	14·7	9	27·3	13·9	16	29·0	14·4
6	27·1	13·0	7	25·3	13·6	5	30·1	13·6	7	25·5	13·0	16	26·7	12·8
7	26·7	12·8	4	24·4	14·2	5	28·1	12·9	6	25·9	12·2	12	27·8	12·9
8	26·2	13·5	6	23·8	13·2	2	29·2	12·6	4	25·6	13·7	11	27·0	14·1
9	24·8	13·4	5	21·4	12·3	1	25·2	14·2	2	25·3	12·5	10	26·7	14·3
10	23·3	12·2	3	21·6	12·2	3	23·4	11·7	4	22·2	13·5	4	26·2	12·7
11	27·5	12·6	1	24·6	14·4	—	—	—	—	—	—	2	31·6	12·9
12	31·6	16·7	—	—	—	—	—	—	—	—	—	—	—	—

[1] Milligrammes per 100 millilitres.

to the age of the infant at the return of the menses. From this it may be seen that in those cases in which menstruation commenced during the first month after the birth of the baby the mean calcium content of the milk was appreciably below the mean values for the whole population and equally below the means for the other classes. The phosphorus values do not differ significantly.

Since this problem does not appear to have been investigated previously, no comment can be offered on these results.

Calcium and Phosphorus Content of the Milk of Women whose Infants Were Found to be Suffering from Rickets.

Earlier investigators, including Bahrdt and Edelstein,[12] Brown *et alii*,[17] Courtney and Brown,[18] and De Buys and von Meysenbug,[5] reported an appreciable lowering of the calcium content of milk from mothers of rhachitic infants; but Telfer[19] in 1924 found no significant differences and stated that rickets might develop in infants ingesting a milk of high calcium content. This is in agreement with the findings in several cases studied in this investigation, and reference to Table X will show that the difference between the two types of milk with regard to calcium and phosphorus content is slight. This is not surprising in consideration of the statement of Clements[16] that "the percentage absorption of calcium is probably the most important post-natal factor in both the development and the persistence of rickets during the first half of the first year of life". The standard of diagnosis of rickets is according to Clements,[20] whose examinations included the subjects of this investigation. As many of these had already received vitamin *D* as a result of Dr. Clements's findings, the percentage of rhachitic infants was well below fifty.

The Effect of the Administration of Extra Calcium to the Mother on the Calcium Content of the Milk.

In 1937 Leitch[13] appealed for further investigations to confirm the finding by Herz[10] and Toverud[21] that it was possible to modify the calcium content of human milk by giving extra calcium to the mother.

In this series five women with milk of initially low calcium content were selected as suitable subjects to be given extra calcium by mouth.

Undoubtedly milk is the most suitable vehicle with which to provide additional calcium; but in order to ensure that the subjects would consider that they were being "treated" for their deficiency, calcium in the form of calcium lactate was prescribed. Because the lactate portion of the molecule is very large in comparison with the calcium portion, it is necessary to give about 120 grains (two level teaspoonfuls) per day in order to provide calcium equivalent to that present in one pint of milk. Arrangements were made for these subjects to take two level teaspoonfuls per day of calcium lactate; this was provided for them.

As adequate vitamin *D* is necessary for optimal absorption[13] of calcium from the alimentary canal, three of the patients were also given a halibut liver oil concentrate containing vitamins *A* and *D*. The dose was about 1,200 international units per day. The results of this medication is given in Table XI.

It is admitted that the number of subjects treated in this way was small, and in one case at least (Case IV) the treatment was not continued long enough to enable any conclusion to be drawn.

In Case I, in which calcium lactate alone was given, some small elevation of the calcium content of the milk was observed; but this was well within the normal range of variation seen in the other cases in this series.

TABLE X.
Showing the Calcium and Phosphorus Content of the Milk of Mothers of Rhachitic and Non-Rhachitic Infants during Lactation.

Age of Infant. (Months.)	Rhachitic (32 Cases).				Non-Rhachitic (124 Cases).			
	Number of Cases.	Calcium. (Milligrammes per 100 Millilitres.)	Phosphorus. (Milligrammes per 100 Millilitres.)	Calcium/ Phosphorus Ratio.	Number of Cases.	Calcium. (Milligrammes per 100 Millilitres.)	Phosphorus. (Milligrammes per 100 Millilitres.)	Calcium/ Phosphorus Ratio.
1	1	20·9	13·6	1·5	16	27·2	16·0	1·7
2	5	26·0	13·8	1·9	46	29·6	14·9	2·0
3	5	25·6	12·4	2·1	42	30·0	14·6	2·1
4	13	27·9	12·5	2·2	37	29·6	13·6	2·2
5	14	27·2	13·6	2·0	32	28·0	13·7	2·0
6	6	27·5	13·6	2·0	29	26·7	13·4	2·0
7	7	26·9	14·1	1·9	27	26·6	12·8	2·1
8	3	28·5	14·1	2·0	18	25·3	13·1	1·9
9	4	28·7	14·4	2·0	16	24·6	13·5	1·8
10	1	20·1	13·2	1·5	10	23·0	12·8	1·8
11	—	—	—	—	2	27·5	12·1	2·3
12	1	31·6	16·7	1·9	—	—	—	—

TABLE XI.

Effect of Extra Calcium and Vitamin D on Calcium Content of Maternal Milk.

Time Relationship of Sample to Medication.	Case I. Calcium Lactate.		Case II. Calcium Lactate.		Case III. Calcium Lactate *plus* Vitamin *D*.		Case IV. Calcium Lactate *plus* Vitamin *D*.		Case V. Calcium Lactate *plus* Vitamin *D*.	
	Age of Infant.[1]	Calcium Content.[2]	Age of Infant.	Calcium Content.	Age of Infant.	Calcium Content.	Age of Infant.	Calcium Content.	Age of Infant.	Calcium Content.
Before medication	6	24·4	14 15 18	26·5 25·6 25·6	6	20·9	3 5 6	19·6 18·9 17·4	5	18·5
Commencement of medication	14		20		4		6		7	
During medication	15 20	25·6 24·2	21 25	23·3 24·6	6½ 7 10	25·5 23·0 28·1	8	24·0[3]	8 11 14 16 17 19	23·9[4] 24·7[4] 24·6[4] 20·5 21·2 19·3
Cessation of medication	23		26		12		8		26	
After medication	26 30 32 36 40	22·7 22·3 21·3 21·3 22·9	35 39	22·7 22·0	14 16 17	22·3 22·5 21·4			30 33 38	18·1 18·7 18·7

[1] Age in weeks.
[2] Calcium content of maternal milk in milligrammes per 100 millilitres.
[3] The infant in Case IV was suddenly weaned shortly after this test was made.
[4] During this period the subject in Case V was taking calcium and Vitamin *D*; but later she omitted the Vitamin *D* because she objected to the taste.

In the two cases in which calcium lactate and vitamin *D* were given, a rise in the calcium content of the milk occurred. In Case III the mean rise was 4·6 milligrammes *per centum*, or an increase of 22%. In Case V the mean rise was 4·4 milligrammes *per centum*, or an increase of 22%; the value dropped again after the subject ceased taking vitamin *D* and the mean value of three readings during the time she was taking calcium lactate alone was almost the same as the premedication level. It is to be noted that the infants in these two cases exhibited clear signs of rickets, whereas those in the other three cases did not.

It is not possible to make any generalizations on the results of this experiment; but from the limited number of cases in the series it would seem that the addition of calcium alone to the mother's diet does not greatly influence the calcium content of the milk. If, however, the calcium therapy is accompanied by vitamin *D* medication, the calcium content of the milk can be elevated about 22%. It is important to notice that in none of these cases were high values obtained, and that the elevated calcium content did not even reach the mean value of the whole series.

Toverud[(21)] found that the calcium content of milk could be raised by 25% by the administration of extra calcium in the diet. I was not able to ascertain whether this worker had incorporated vitamin *D* with the calcium.

Summary.

1. The calcium and phosphorus content of the breast milk of 165 Australian women was found to agree fairly closely with that of European and American women, and ranged from 17 to 42 milligrammes *per centum* and from 7 to 21 milligrammes *per centum* respectively.

2. The mean calcium and phosphorus values varied according to the period of lactation, and individual samples of milk followed this general trend. It was, therefore, not possible to give an average value for any sample of milk.

3. Calcium values reached their peak between the second and fourth months and then steadily declined until the tenth month.

4. Phosphorus values fell steadily from a peak at the first month until the eighth month, when they temporarily rose, and fell again after the ninth month.

5. The calcium:phosphorus ratio was mostly constant throughout the period of lactation.

6. The influence of the season of the year on calcium content of maternal milk was investigated, but the number of cases was too few to confirm any findings.

7. The calcium and phosphorus content of milk in respect to the number of previous children did not appear to vary greatly.

8. The early onset of menstruation after parturition was considered as a possible factor in the lowering of calcium and phosphorus values of the milk.

9. The mean calcium and phosphorus content of the milk of mothers of rhachitic infants was not significantly different from that of mothers of normal infants.

10. The effect of the administration of extra calcium to the mother in raising the calcium level of the milk was studied in five cases and found to be noticeable only in the two cases in which vitamin *D* was administered concurrently with the calcium.

References.

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RICKETS IN INFANTS AGED UNDER ONE YEAR: THE INCIDENCE IN AN AUSTRALIAN COMMUNITY AND A CONSIDERATION OF THE ÆTIOLOGICAL FACTORS.

By F. W. CLEMENTS,

Australian Institute of Anatomy, Canberra.

PREAMBLE.

IN 1932 Maddox[1] showed that 52% of the infants attending the out-patient department of an infants' hospital in Sydney had unmistakable signs of rickets. In 1938 Clements[2] reported that out of 255 infants and children between the ages of nine months and two years examined by him, 60 showed the accepted signs of active rickets. For many years rickets has been attributed, in part at least, to an inadequate supply of vitamin *D* within the body of the affected infant. This vitamin *D* can be supplied in food or in medicinal form or by exposure of the infant's body to sunlight. It has been argued that as Australia is richly provided with sunshine, the percentage of rickets in Australia should be very low.

In 1938 a long-term investigation was planned which had as its first aim a survey of a group of infants to determine the incidence of rickets, and then if rickets was found to occur, to endeavour to make a contribution toward the ætiology of the disease.

The Source of the Material.

The infant welfare centres of the Canberra Mothercraft Society were made available for this investigation. The clientele of the centres is representative of an average Australian population, except that the very poor are not found there and the percentage of underprivileged is small.

The Definition of Rickets.

Drummond and Wilbraham[3] believe that evidence is available which demonstrates that severe rickets was extremely common in England in the seventeenth century, but that the disease reached its worst form during the intense industrialization of the forties of the nineteenth century. This type of rickets, with its accompanying extreme bony deformities, became the standard picture of the rickets described in text-books of medicine and pædiatrics which has persisted to this day. In a recent text-book on diseases of infants and children,[4] the following description of rickets is given:

> Amongst the earliest manifestations are head swelling, restlessness, rocking of the head on the pillow . . . In a short time beading of the ribs is noticed in most cases, other bony enlargements now develop, especially of the skull, ribs, wrist and ankles, whilst characteristic alterations of the shape of the chest appear and the abdomen becomes pot-bellied. The fontanelle fails to close at the proper time, growth of the body is interfered with, curvature of the long bones and spine is produced.

This is a picture of florid rickets, a condition that is rare in Australia.

Within the last decade rickets has become recognized as defective calcium metabolism made manifest by imperfect calcification, even to a minor degree, of the growing ends of long bones. This means that it is possible for the condition to exist in the absence of any obvious clinical manifestations. It also means that in the early stages the diagnosis of the disease is possible only by means other than clinical.

In many parts of the world surveys have shown that mild rickets is common in infants and young children. Moore and his collaborators[5] stated that over 90% of 900 children aged from five to six and a half years in two American cities had signs of past or present rickets. In South America Carrau and Bazzano[6] have stated that rickets is present in 15% to 18% of the infants in the hospitals of Montevideo. It is extremely difficult to compare the incidence of the disease in one country with that in another, for the standards by which the condition is diagnosed vary and are oft-times not stated by the workers.

Undoubtedly the trend of world opinion is that the florid rickets of the nineteenth century is fast disappearing, but that a high percentage of mild rickets still persists.

The Diagnosis of Rickets.

The child who is suffering from florid or advanced rickets presents a picture easily recognized by the classical text-book description, so that further aids to diagnosis are unnecessary and a radiological examination of the long bones merely serves to confirm the clinical diagnosis. The recognition of either the early stages or the milder forms is more difficult, and recourse to non-clinical methods is necessary. Two such methods are available—the radiological examination of the growing ends of long bones and the analysis of blood serum for calcium, phosphorus and phosphatase.

Radiological Examination of Long Bones.

A Swedish school of radiologists, headed by Lindblom,[7] has described a degree of decalcification of the metaphysis as the earliest signs of rickets. Whilst it is possible that these early signs are characteristic of rickets, I have followed an American worker[8] and have used the standards for radiographic diagnosis of rickets that have been set by Eliot and Park.[9] The significance of the radiographic changes described by Lindblom will be discussed when the ætiological factors responsible for rickets are considered.

Serological Diagnosis of Rickets.

Howland and Kramer[10] in 1921 held that the level of inorganic phosphorus in the blood serum was lower in rhachitic than in normal infants and that in uncomplicated rickets the serum calcium content was normal. It has since been shown that rickets can occur when neither of the above conditions exists. The serum inorganic

phosphorus level usually falls in rickets, but not with sufficient consistency to be a diagnostic feature.

Robison[11] and Robison and Soames[12] drew attention to the importance of blood and bone phosphatase content in rickets. Although the serum phosphatase content is increased in a number of bone diseases, it is nevertheless of value as a diagnostic sign of rickets.

Because of the obvious difficulties associated with the collection of blood, serum phosphatase estimations were not employed in this survey.

Clinical Diagnosis of Rickets.

As Jeans[13] has observed, the clinical diagnosis of mild rickets in the absence of positive radiographic evidence is open to serious doubt, and in this investigation mild rickets has been diagnosed only on radiographic evidence. Every infant was carefully examined at each visit for the presence of those clinical features usually considered to be associated with rickets. The presence of any sign was recorded on the history sheet, to be subsequently compared with the radiological findings.

This Investigation.

The Technique Employed.

During the past two and a half years nearly 500 infants have been seen and examined radiographically for rickets. Of these, 150 have had radiographs taken regularly each month throughout the first year of life. Many infants were examined regularly, but for shorter periods, and whilst the records of these cases have not been included in the main results, many of the cases have been reported in separate sections; particularly does this apply to the relationship between the calcium content of maternal milk and the development of rickets.

The initial examination and radiograph were made as soon as possible after the discharge of the mother from hospital, and usually took place toward the end of the first month. Thereafter, although the mother attended the mothercraft centre regularly at intervals of approximately one week, the infant was reexamined and a radiograph of the right wrist was taken once a month. In those cases in which moderate rickets developed, radiographs were frequently made at shorter intervals.

The Standard by which Rickets was Diagnosed.

A child was considered to have suffered from rickets if one radiograph was obtained showing definite rhachitic changes. Many single radiographs were obtained in which the growing end of the ulna was not normal and showed slight irregularities; at the time these radiographs were labelled "doubtful". At the next radiograph in the series some of these babies showed frank mild rickets, whilst the condition of others had returned to normal. The ultimate classification of the case was determined by the final radiological picture. The "doubtful" appearances that became normal were classified as "normal". It is considered that infants that had "doubtful" radiographs were suffering from incipient or borderline mild rickets, their condition, because of therapeutic or other measures, having returned to normal before the definite radiological signs appeared.

The Incidence of Rickets amongst 134 Full-Term Infants of whom Radiographs had been obtained regularly throughout the First Year of Life.

Of the 134 full-term infants in this series, of whom radiographs had been obtained regularly each month throughout the first year of life, 63, or 47%, had radiographic evidence of definite mild rickets at some period during the first year of life. Thus the actual incidence of radiographic rickets agrees closely with the findings of Maddox.[1] In every case the changes were of the mild type, and as will be seen from the later stages of this work, the duration was very short in a high percentage of instances.

The Incidence of Rickets amongst Eight Premature Infants.

The criterion of prematurity set down by Griffith and Mitchell[14] is a birth weight of less than 2,500 grammes (5·5 pounds) or a length of under 45 centimetres (17·7 inches). Accurate measurements of the birth length of each infant were not available and prematurity was determined on weight alone. Judged by this standard, eight infants in our series were premature. Without exception all these infants developed rickets. A list of the infants and the salient points in their histories are given in Table I.

In three cases medicinal treatment with vitamin *D* was commenced before the rickets appeared, and in two of these cases the rickets lasted but two months. In all cases in which continuous radiographic records were obtained, the rickets developed at about three months of age.

It is recalled that Hamilton[15] and Muhl[16] found rickets to occur in almost every premature infant, thus agreeing with our findings.

The significance of these results will be discussed later.

The Incidence of Rickets amongst Twins.

Three sets of dissimilar twins occurred in our series. Four of the infants developed radiographically proved rickets before the sixteenth week of life, as the details in Table II show.

TABLE II.
Salient Points in the History of Three Sets of Twins.

Set.	Sex.	Birth Weight.	Age of Onset of Rickets. (Weeks.)	Age when Cured. (Weeks.)
A	Male.	5 lbs. 3 ozs.	14	21
	Female.	4 lbs. 6 ozs.	14	21
B	Female.	6 lbs. 3 ozs.	15	24
	Female.	4 lbs. 9 ozs.	9	24
C	Male.	5 lbs. 0 oz.	Nil.	—
	Female.	4 lbs. 15 ozs.	Nil.	—

It is interesting to note that the smaller twin of set B developed rickets before her heavier, more mature sister. Set C is interesting, for when it was discovered by radiographic examination that the pregnancy was a twin one,

TABLE I.
Premature Infants.

Case Number.	Birth Weight.	Type of Feeding.	Vitamin *D*.		Age at Onset of Rickets. (Weeks.)	Duration of Rickets. (Months.)
			Given.	Age Treatment Started. (Weeks.)		
423	3 lbs. 0 oz.	Breast.	Yes.	2	14	2
431	3 lbs. 8 ozs.	Breast and "Vi-Lactogen".	As "Vi-Lactogen".	10	15[1]	2
53	4 lbs. 0 oz.	Cow's milk.	Yes.	6	8	4
139	4 lbs. 6 ozs.	Breast.	Yes.	23	14[1]	4
99	4 lbs. 14 ozs.	Breast.	Yes.	10	10	1
27	5 lbs. 0 oz.	Condensed milk and cow's milk.	No.	—	12	6 at least[2]
8	5 lbs. 0 oz.	Breast.	Yes.	21	10	5
422	5 lbs. 4 ozs.	Breast.	Yes.	5	14	2

[1] First radiograph in the series obtained at this age; child may have had rickets before this age.
[2] Child left district at age of seven months.

the mother was advised to drink milk freely, and she actually consumed one quart of milk per day during the last two months; in addition she took a vitamin *D* preparation, which provided about 1,500 international units of vitamin *D* per day.

The Age of Onset of Rickets.

The age of the infant at which the first radiograph revealing rickets was obtained varied from one month to eight months. The distribution of the 63 cases of rickets amongst the full-time infants in relation to age of onset is shown in Table III.

Table III.
Showing Age of Onset of Rickets.

Age in Lunar Months.	Number of Infants with Onset at this Age.
1	1
2	11
3	23
4	16
5	6
6	4
7	1
8	1

It may be seen that in 80% of the cases the onset occurred before the sixteenth week. A small group of infants developed rickets in the second half of the first year.

The Duration of Rickets.

The duration of the rickets in those cases in which it developed during the first sixteen weeks of life is shown in Table IV.

Table IV.
Duration of Rickets.

Duration in Months.	Number of Cases.
1	6
2	10
3	9
4	6
5	8
6	6
7	3
8	3

The contents of Table IV, although interesting, are not informative, for, as will be shown later, the duration of most cases of rickets is subject to conditions over which medical control can be exercised.

Discussion.

The results of this investigation have demonstrated that mild rickets is not uncommon amongst infants aged under one year. For a full appreciation of the significance of these findings it is necessary to analyse the factors responsible for the development of rickets and for their duration, and to endeavour to measure the influence of mild rickets, detectable only by radiographic means, on the general growth and development of the infant.

The Influence of the Ante-Partum Diet of the Mother on the Development of Rickets in the Infant.

In the fœtus bone develops in preexisting cartilage by the continual deposition of calcium salts. The writings of Coons and Blunt[21] and of Hamilton[23] have shown that nearly three times as much calcium is deposited in the fœtus in the last two months of pregnancy as is deposited during the preceding seven months, for a seven-months fœtus contains about 8·0 grammes of calcium and a full-term infant about 30·0 grammes. Opinions as to the behaviour of the calcium and phosphorus during pregnancy vary. Standard text-books on obstetrics frequently contain a statement similar to the following: "Where the food supply of the mother does not contain an adequate amount of calcium for the fœtus the calcium salts are removed from the maternal skeleton." I have been unable to trace the scientific data upon which statements of this type are based.

Likewise, articles on dental caries in pregnancy not infrequently contain opinions similar to the following, taken from a recent work: "In pregnancy and lactation teeth often become tender and carious. The cause is undoubtedly the drain of calcium by the fœtus . . . acting on the cementum and teeth." Both Klein[19] and Schour,[19] in recent reviews of the literature on experiments with both animals and human beings, are unconvinced that pregnancy has any effect upon the teeth.

A review of the known facts will be useful at this stage.

The Calcium Content of Maternal and Infantile Skeletons.—McCollum and his co-workers[20] stated that the adult skeleton contains from 1,400 to 2,000 grammes of calcium, or about 2% of the body weight. On these figures the amount of calcium in the body of the average woman (120 pounds) would be about 1,100 grammes. A full-term fœtus contains from 25 to 30 grammes of calcium, or between 2·5% and 3·0% of the total calcium in the mother's skeleton. From 16 to 22 grammes of this calcium are obtained by the fœtus during the last three months of gestation. This means that even at the maximum figure the fœtus requires about 0·6 gramme per day. Even if the diet were poor in calcium, some of this calcium would be supplied from the food eaten. If the full calcium requirement of the fœtus, namely, 30 grammes, was withdrawn from the mother, it is difficult to conceive of human experiments which would demonstrate a reduction of 3% of calcium content of the skeleton over a period of nine months. Calcium and phosphorus metabolism experiments merely indicate the degree of equilibrium and do not indicate in the case of a negative balance whether the fœtus is withdrawing calcium from the skeleton of the mother or suffering incomplete calcium storage. It is true that osteomalacia and osteoporosis are recognizable by radiographs; but in the former condition the inorganic salts in the bones are reduced by 50%.[21] The literature on the calcium content of the skeleton of newborn infants and its relationship to the ante-partum diet of the mother is sketchy and conflicting. Hamilton[23] collected the results of a number of analyses of the calcium content of 10 full-term infants and showed that calcium content varied from 5·01 grammes per kilogram of body weight to 10·27 grammes, with a mean of 8·127 grammes per kilogram. If the normal full-term fœtus withdrew all the calcium it required from the maternal skeleton, it is reasonable to assume that the calcium content of 10 full-term infants would not have varied by 100%. If, however, surplus calcium for storage in the long bones is obtained from the mother's diet rather than from her skeleton, the divergencies reported by Hamilton would be expected.

Relationship of Fœtal Skeletal Calcification to Maternal Calcium Metabolism.—Maxwell and his co-workers in China[22][23][24][25][26] have collected 17 cases of rickets developing in the infants of women suffering from osteomalacia, establishing beyond doubt the causal relationship between osteomalacia in the mother and rickets in the newborn infant. Osteomalacia is essentially a disease due to vitamin *D* deficiency and calcium shortage. The mineral content of the skeleton is greatly reduced, even to 50% of the normal; but even with this great reduction relatively large quantities of calcium remain in the skeleton from which the fœtus could obtain its supplies if this was the regular route by which it obtained all its calcium. Instead, it would seem that the calcium deficiency in the diet of the mother that was associated with the development of the osteomalacia also played a part in the development of the calcium deficiency in the skeleton of the fœtus. Strong support for this reasoning is found in the cases of osteomalacia studied by Liu and his co-workers.[27] This investigation, which has yielded some important findings, consisted of a study of the calcium and phosphorus metabolism of the mother and the infant concurrently. In one case the mother had had advanced osteomalacia, but during the course of the pregnancy she

received approximately two grammes of calcium per day with large doses of vitamin *D*. During the last four months of gestation 40% to 50% of calcium ingested was retained. In the words of the Chinese workers: "This enabled her not only to meet the fœtal requirements, but also to replenish her own skeletal store." The baby, delivered at full term by Cæsarean section, was "normal in every respect". Another case in this same series presented the opposite picture. A woman was found to be suffering from osteomalacia on the occasion of the birth of her eighth infant, which shortly after birth developed rickets. In this case the mother had received no extra calcium or vitamin *D* during the pregnancy. Booher and Hansmann[28] determined the calcium content of the tibiæ of a number of full-term infants which died shortly after birth. They failed to find any correlation between the calcium and vitamin *D* contents of the diet of the mother during the pregnancy and the calcium content of the bones examined. As the calcium intake and vitamin *D* consumption of the eight mothers investigated varied widely, some correspondence might have been expected. This is the only piece of work of this type in the literature, and in view of its importance in studies of this kind it should be repeated. Coons and Blunt[17] concluded from the radiographic examination of the bones of normal infants from well-fed mothers that the calcification of the bones was best when the retention of calcium and phosphorus by the mother during pregnancy was highest. Toverud[29] has reported early rickets in infants born of mothers whose diets had been poor in calcium.

The Mechanism of Fœtal Calcium Acquisition.—In 1922 Hamilton[15] advanced the hypothesis that the long bones of the fœtus acted as a reservoir for calcium, which was stored during the last months of intrauterine life. Eliot and Park[9] have expressed a similar opinion. Support for this hypothesis is found in another direction; Eliot and Park[9] and Le Gros Clark[20] have both stated that at birth the shaft of long bones consists of a closely meshed core of osseous trabeculæ and the formation of the marrow cavity takes place some time after birth. Lindblom[17] has described the radiographic appearances of minute changes of the growing ends of long bones commencing as early as the third day after birth. The early radiographic signs start as a decalcification of the bony tissue immediately beneath the calcifying zone of the diaphysis—that is, the metaphysis. In addition, Lindblom has recorded a general decalcification of the bone appearing as a thinning of the spongiosa columns and the cortex. It was observed by this worker that the administration of vitamin *D* had no effect upon this process of decalcification, since infants treated showed no deviation from those not treated. These changes were noted from the third day onward and were due to a decalcification rather than to a failure of calcification such as occurs in rickets. Lindblom strongly affirmed that these decalcification processes were the earlier radiographic signs of rickets. Those cases discussed in the present paper, in which regular radiographs were obtained, have been reviewed each month from the first to the fifth inclusive. There were 31 infants in this series. Four of them developed rickets without any of the signs described by Lindblom being recorded. Five infants presented normal radiographic appearances throughout the first five months. Twenty-two infants showed metaphysial decalcification, general decalcification or sclerosis—the signs described by Lindblom—and 12 of these developed rickets. In the remaining 10 the decalcification gave way to normal calcification without the appearance of rhachitic changes. The details of these cases will be referred to later; at this stage it is relevant to observe that this series of radiographs demonstrated a range of calcification in long bones extending from normally progressive calcification at one end through mild decalcification processes (which were arrested before the onset of rhachitic changes) to those cases in which frank rickets developed at the end of the decalcification process. These results strongly suggest that the decalcification observed by both Lindblom and myself is the lower limit of the physiological process of calcium withdrawal from its normal storehouse. Should the hypothesis advanced by Hamilton prove to be a rule of bone metabolism, then the whole conception of the calcium requirements of the young infant will require revision.

A Consideration of the Possible Requirements of Young Infants.—The average calcium content of 10 full-term infants in Hamilton's series was 8·127 grammes per kilogram of body weight. By the use of a weight chart developed from the weights of Australian infants,[31] it has been possible to determine the amount of calcium that an Australian infant of average weight would require to absorb in order to maintain the calcium content of the skeleton at the same level as at birth. These amounts are given in Table V. Leitch[22] has worked out similar figures using

TABLE V.

Daily Calcium Requirements of an Infant at Varying Ages Necessary to Maintain the Same Calcium Concentration as at Birth.

Age in Weeks.	Infant's Weight in Grammes.	Total Amount of Calcium in Body at 8·127 Grammes per Kilogram. (Grammes.)	Daily Requirement of Calcium During the Month Necessary to Maintain Calcium Level at Birth. (Grammes.)
Birth	3,688	29·97	0·147
4	4,195	34·09	0·214
8	4,932	40·07	0·200
12	5,613	45·6	0·172
16	6,208	50·4	0·164
20	6,775	55·0	0·157
24	7,314	59·4	

English weight charts. The possibility that infants may receive this amount of calcium can be explored from available Australian data. Ritchie[23] has shown that the calcium content of Australian maternal milk has a range from 17 to 42 milligrammes per 100 millilitres with a mean of nearly 30 milligrammes. Wake[34] has demonstrated in breast-fed infants that the absorption of calcium from the intestinal tract varies from 10% to 70% of the calcium ingested, with a mean at 45%. By a combination of the findings of these two workers, the range of possible absorption at different levels of intake has been worked out and the results are given in Table VI. Three levels of calcium content of maternal milk have been taken arbitrarily: low, 22 milligrammes per 100 millilitres; average, 30 milligrammes per 100 millilitres; high, 36 milligrammes per 100 millilitres. Three levels of absorption have been selected: low, 30%; average, 45%; high, 60%. When the conditions are such that the absorption of calcium reaches the daily requirements, the figures have been shown in italics. If we work upon the thesis that the infant obtains all its post-natal requirements of calcium from its post-natal diet, it will be seen that not until the fourth month, when the intake is approaching maximum figures and the actual requirement is falling, does it appear that the daily acquisition of calcium is equal to that necessary to maintain the calcium content of the skeleton at the same concentration as that present at birth. In these circumstances it is highly improbable that in the first three months of life any but a small percentage of infants will retain sufficient calcium to maintain the birth concentration. The regularity with which premature infants develop rickets suggests that the deposition of calcium toward the end of intrauterine life plays an important part in the post-natal calcification of the growing ends of bones. If post-natal calcification depended entirely upon post-natal calcium supply, then it should be possible in at least a few cases to control the development of rickets in premature infants. It was the experience of Hamilton[15] and Muhl[26] as well as of myself that rickets will develop in premature infants despite all steps taken to prevent it. Very little work has been carried out on skeletal weights of infants of various ages, for the only reference I could find was to unpublished work by Venar and Todd, quoted in "The White House Conference" series.[33] With respect to the values for wet weights of infants from birth to twelve months, I do not agree with the deduction drawn by Leitch[22] that the fresh weight of the skeleton increased in proportion to body weight. The rate of skeletal growth is much more rapid during the first two months of life

TABLE VI.

Calcium Absorptions at Different Levels of Calcium Content of Human Milk at Various Ages.

Age of Infant.	Average Daily Milk Intake. (Cubic Centimetres.)	Percentage Absorption of Calcium from Intestinal Tract.	Calcium Content of Milk. Low: 22 Milligrammes per 100 Millilitres.	Calcium Content of Milk. Medium: 30 Milligrammes per 100 Millilitres.	Calcium Content of Milk. High: 36 Milligrammes per 100 Millilitres.	Average Daily Calcium Requirements Necessary to Maintain Calcium Concentration in Body Same as at Birth (from Table V). (Milligrammes.)
At end of 4th week	770	30 45 60	51 76 102	69 104 140	83 125 *167*	147
At end of 8th week	900	30 45 60	59 89 119	82 122 162	97 146 192	214
At end of 12th week.	1,000	30 45 60	66 99 132	90 135 180	108 162 *216*	200
At end of 16th week.	1,070	30 45 60	70 106 141	93 140 *187*	116 *173* *230*	172
At end of 20th week.	1,130	30 45 60	74 112 150	102 150 *200*	122 *184* *245*	164
At end of 24th week.	1,200	30 45 60	79 119 *158*	108 *162* *216*	130 *195* *260*	157

than at any other stage; yet during this period the actual calcium intake is small because of the relatively low milk intake. The rapid increase in the wet skeletal weight during the first two months after birth could be achieved by a heavy deposition of calcium or by a reorientation of the water distribution, or by both. It is recalled that at birth the shafts of long bones are closely packed with cancellous tissue, much of which is removed soon after birth to be replaced by the characteristic marrow cavity. Here we have water and fat replacing bone and the bony tissue being then available for the calcification of new bone. Studies of bone density should prove this hypothesis; but the only figures for bone density are in unpublished work by Venar and Todd, and in their series no data are given of the history of the infants from which the bones were obtained. Some proof of the changes in bone after birth can be obtained from serial radiographs. Barber[35] has demonstrated that osteoporosis in fracture cases is limited to absorption of the finer secondary trabeculæ. In a radiograph these finer secondary trabeculæ produce a cloudy appearance between the coarser architectural trabeculæ of normal bone.[36] In almost all cases in our series in which a radiograph was obtained in the first two weeks of life, it had that cloudy appearance completely fogging the primary trabeculæ. Within two or sometimes three months the radiographic appearance cleared, leaving the well defined secondary trabeculæ. It has frequently been recorded[36] that in rickets absorption of the secondary trabeculæ occurs. From our series of radiographs it would seem certain that absorption preceded the actual appearance of rickets. Francis[38] made the discovery that bone already laid down in ossification centres might be reabsorbed as a result of rickets. This is evidence of the withdrawal of calcium from already calcified tissue to help in the calcification of new bone.

Recapitulation.—Enough evidence has been produced at least to lend support to the hypothesis that the skeleton of the fœtus carries a supply of calcium for the post-natal use of the infant in skeleton building. If this is so, then it will mean that the daily calcium requirement of the infant in the first two or three months after birth will be less than the amounts shown in Table V. It will also mean that a higher percentage of infants will obtain the daily requirements of calcium than is indicated by Table VI. As I shall show from this series, this is undoubtedly the case; it will also be demonstrated later that the development of rickets bears a distinct relationship to the opportunities for calcium storage within the infant enjoyed by the mother during pregnancy.

Review of Results in the Light of the Preceding Discussion.—As part of this investigation the food intake of the mother during her pregnancy has been recorded. The main source of calcium in any diet is the milk consumed. In an analysis of the average Australian diet, it was recently shown[37] that when the milk consumption was half a pint per day it provided nearly 50% of the total calcium intake when the latter was about 0·5 gramme per day. Toverud[39] and the League of Nations Technical Commission[39] recommend one litre (one and a half pints) of milk as the amount necessary to provide the calcium requirements for pregnant women. The present inquiry was limited to the amount of milk consumed during pregnancy, especially during the last two months. The quantity of vitamin *D* within the body largely influences the amount of calcium absorbed from the alimentary tract. As the vitamin *D* obtained from exposure to the sun is extremely difficult to measure, this inquiry included only the consumption of medicinal vitamin *D*. One of the medical practitioners in Canberra, Dr. J. A. James, has for some time past prescribed for pregnant women in his care a preparation containing vitamins *A* and *D* in concentrated form. It is his custom to order 100 capsules at about mid-term with instructions to take one capsule per day. This would supply about 1,700 international units of vitamin *D* per day. Adequate milk intake by the mother combined with the consumption of vitamin *D* should ensure adequate calcium absorption making available adequate calcium for the fœtus. I have correlated the development of rickets during the first four months with the amount of milk consumed and the vitamin *D* intake of the mother during the last two months of gestation. The results are given in Table VII.

TABLE VII.

Showing the Number of Mothers who Consumed Various Quantities of Milk and Vitamin D and the Number of Cases of Rickets that Developed in Their Offspring in the First Sixteen Weeks of Life.

Approximate Amount of Milk Consumed per Day by Mother in Last Two Months of Pregnancy.	Mother did Not Take Medicinal Vitamin *D*.				Mother Took Medicinal Vitamin *D*.			
	Number of Cases.	Rickets in Infants.			Number of Cases.	Rickets in Infants.		
		Number.	Subtotal.			Number.	Subtotal.	
			Number.	Percentage.			Number.	Percentage.
Nil	20	11	18	50	10	4	8	50
Less than half a pint regularly or more regularly.	16	7			6	4		
One-half to one pint per day	11	3	15	37·5	8	1	3	13
One pint or more per day	28	12			15	2		

Four facts emerge from Table VII. (i) The outstanding observation is the high percentage of mothers who did not drink milk during their pregnancy. This observation is not confined to Canberra, for during the last twelve months I have had the opportunity of interrogating mothercraft nurses in all parts of Australia and the story is universal. The reason is not an economic one, for the failure to drink milk during pregnancy is met at all social levels in the community. In many cases it is due to a lack of an appreciation of the need for the extra calcium in this form; in another large group of cases it is due to an absolute aversion to milk. I am sure that this could be overcome if women used some of the many methods available for flavouring milk. (ii) The apparent ineffectiveness of medicinal vitamin *D* in the absence of an adequate calcium intake is obvious. Vitamin *D* aids the absorption of calcium from the alimentary canal. (iii) The improvement in the incidence of rickets when the milk intake was adequate, even in the absence of medicinal vitamin *D*, is demonstrated. The beneficial effects of sunlight as a source of vitamin *D* cannot be overlooked in these cases. (iv) The outstanding feature of the table is the low incidence of rickets among the infants whose mothers consumed a reasonable quantity of milk and had received vitamin *D*. In 31 cases the first radiograph was taken as early as the second week of life, and thereafter in these cases regular radiographs were taken at intervals of exactly four weeks. The radiographs were collated with the calcium consumption and vitamin *D* intake of the mothers during the last months of pregnancy. The results are shown in Table VIII.

The results given in Table VIII show that the degree of decalcification of the long bones of the infant bears a direct correspondence to the type of the mother's ante-partum feeding. It would seem that when opportunities existed for calcium storage obvious decalcification did not take place, whereas in those cases in which the ante-partum diet of the mother did not permit of calcium storage rickets developed early in post-natal life. The other important fact presented in Table VIII is the effect of the early administration of medicinal vitamin *D* to the infants. This will be enlarged upon in the next section.

Summary.

1. Indirect evidence has been brought forward to support the suggestion that the fœtal skeleton acts as a reservoir for the calcium required during the early months of life.

2. Whilst a certain basic quantity of calcium necessary to lay the foundation of the fœtal skeleton may, in the complete absence of calcium from the diet, be withdrawn from the maternal skeleton, extra calcium for storage in the fœtal skeleton is apparently supplied from that present in the mother's food.

3. The storage of calcium by the fœtus seems to determine the immediate post-partum ossification mechanism of the infant. A maternal diet rich in calcium and vitamin *D* seems to protect the infant from rickets during the first half of the year. Poor calcium and poor vitamin *D* intakes by the mother, especially the former, are associated in a high percentage of cases, in which rickets develops early in the infant's post-natal life.

TABLE VIII.

Relationship of Radiographic Changes in Infants to Ante-partum Diet of Mother.

Description of Radiographs.	Number of Cases.	Ante-partum Diet of Mother.			
		Little or No Milk and No Vitamin *D*.	More than Half a Pint of Milk but No Vitamin *D*.	Little or No Milk, but Vitamin *D*.	More than Half a Pint of Milk and Vitamin *D*.
Each radiograph normal one to five months	5	—	3	—	2
Frank rickets either first or second month	4	3	—	1	—
Decalcification, later becoming normal..	12	4 [4][1]	—	3 [1]	5 [1]
Decalcification leading to rickets ..	10	1	5	3 [1]	—

[1] The figures in brackets refer to the number of infants in each series who received medicinal vitamin *D* from the age of one month onward.

4. That this is not the whole story of the ætiology of rickets is clearly demonstrated by the fact that only 50% of the infants born of the women who consumed small quantities of milk developed rickets. If inadequate absorption of calcium by the pregnant woman is the only factor in the development of rickets in the offspring, then one might reasonably have expected all infants in this group to have developed rickets.

The Influence of Post-Natal Feeding on the Development and Persistence of Rickets.

During the first nine or ten months of life an infant may be (*a*) fully breast fed, (*b*) partially breast fed, receiving complementary feeding with some form of cow's milk, (*c*) fully artificially fed with some form of cow's milk. Cow's milk contains about four times as much calcium as human milk, so that even with appropriate modification an infant receiving any portion of its diet in the form of cow's milk must receive more calcium per day than an infant fully breast fed. Considerable work has shown that in ordinary circumstances the percentage of calcium absorbed from cow's milk is less than the percentage absorbed from human milk. Because of these differences it is necessary to consider breast-fed infants separately from artificially fed infants.

In this series, of the 51 infants who developed rickets during the first sixteen weeks of life, 40 were fully breast fed during that time, seven received a mixed diet of breast milk and some form of cow's milk and four were fully artificially fed. It is proposed throughout this section of this paper to confine the remarks to those children who were fully breast fed.

The degree of bone calcification in an infant during the first few months of life will depend upon calcium from two sources. It has been shown in the preceding section that strong evidence exists to support the suggestion that calcium is stored within the fœtal skeleton; this would provide a supply of calcium, for the "remodelling" must provide some calcium. The remainder would come from the food.

Wake[30] has demonstrated that during the first two months of life the percentage retention of calcium varied widely. These variations were associated with both a low calcium intake due to difficulties attendant upon the introduction of breast feeding, maternal milk supply *et cetera*, and also upon a low percentage absorption with a satisfactory intake.

Ritchie[31] has shown that the calcium content of human milk may vary within wide limits. Unfortunately, there is no way short of regular metabolism experiments of assessing the percentage of calcium absorbed.

Throughout this investigation it was possible to compare the calcium content of the maternal milk with the rhachitic history of the infant during the first half of the first year of life. Certain points of correspondence between the calcium content of the maternal milk and the development of rickets emerged. (*a*) Rickets developed in infants of women whose milk contained adequate quantities of calcium. The milk intake of these infants was also adequate. (*b*) The infants of three women whose milk was poor in calcium (less than 20 milligrammes per 100 millilitres) developed rickets. It would seem that, except in cases in which milk has a very low calcium content, the actual calcium intake by the infant does not influence the development of rickets. Undoubtedly the important factor is the total absorption of calcium by the infant.

Numerous workers have shown that the percentage absorption of calcium can be raised and stabilized by the addition of vitamin *D* in some form to the infant's diet. In this investigation it was possible to study the effects of the administration of vitamin *D* upon (*a*) the development and (*b*) the duration of rickets.

The Development of Rickets.

In Table VII the incidence of rickets amongst infants and the amount of milk and vitamin *D* consumed by their mothers are shown. The same case arrangement has been used and the effects of the administration to the infants of vitamin *D*, commenced before the eighth week, upon the development of rickets, has been studied. The vitamin *D* was given either as a 50% emulsion of cod liver oil or as halibut liver oil. The dosage ranged from 400 to 1,000 international units per day. These results are shown in Table IX.

The outstanding facts in Table IX are as follows:

1. The high percentage of infants free from rickets who were given vitamin *D* by mouth within the first two months of life.

2. The influence of vitamin *D* medication upon the infant is especially noticeable in those groups in which the mothers' consumption of milk and vitamin *D*, particularly the former, were unsatisfactory.

3. The contrast in the first group of the table between the percentage of infants who had received no vitamin *D* and had developed rickets (16·8%) and the percentage who had received vitamin *D* and had not developed rickets (66%) is worthy of note.

The conclusion to be drawn from this table is that in those cases in which the diet of the mother has not contained ample calcium and vitamin *D*, early administration of vitamin *D* to the infant will ensure that a percentage, at least, of the children will escape rickets.

This procedure is not completely protective, for five of the infants in this series were given vitamin *D* by mouth early in post-natal life (within two weeks of birth), and yet they developed rickets. There may be two explanations for this state of affairs: (*a*) that the vitamin *D* did not sufficiently raise the percentage absorption of calcium; (*b*) that in the terms of the hypothesis advanced in the first section of this discussion, these children had inadequate ante-natal calcium storage; it was so deficient that it could not be rectified by an adequate calcium intake early in post-natal life. I am inclined to the second explanation. The duration of the rickets in these cases (to be referred to later) supports this belief.

TABLE IX.

The Effects of Administration of Vitamin D to Infants upon the Subsequent Development of Rickets.

Milk and Vitamin *D* Consumption of Mother.		Number of Infants.	Infants who Developed Rickets.			Infants who were Free from Rickets.		
			Number.	Infants who had No Vitamin *D*.		Number.	Infants who Had Vitamin *D*.	
				Number.	Percentage.		Number.	Percentage.
No Vitamin *D*.	Little or no milk ..	36	18	3	16·8	18	12	66
	More than half a pint of milk	39	15	2	13·3	24	12	50
Vitamin *D*.	Little or no milk ..	16	8	2	25·0	8	4	50
	More than half a pint of milk	23	3	—	—	20	2	10

The Duration of Rickets.

Of the 40 breast-fed infants in this series who developed rickets, three did not receive vitamin *D* at any time during their first year, but in one case the mother received 2,000 international units of vitamin *D* per day. In the two cases in which vitamin *D* therapy was not given healing occurred in four and six months, whilst in the third case healing occurred within three months (that is to say, two "Positive" radiographs were obtained) of the time the mother commenced to take the vitamin.

In eight other cases the rickets underwent spontaneous healing without the administration of vitamin *D*, although it was given to the infants after healing was completed. With one exception the cure took place in the summer months and the duration of the rickets varied from one to six months. In four cases the onset was observed in the winter months and the condition persisted in a mild form until the next summer, when it underwent spontaneous cure.

In those cases in which vitamin *D* was administered to the infant the duration of the rickets bore a definite relationship to the point at which the vitamin *D* therapy was instituted. It is important to emphasize the fact that in five cases in which vitamin *D* administration was commenced early in the first month, even though rickets developed, it lasted but one month in four cases and two months in the others.

The duration of rickets in those cases in which the administration of the vitamin *D* followed the diagnosis of rickets is shown in Table X.

TABLE X.

The Duration of Rickets after the Commencement of Vitamin D Therapy.

Duration of Rickets after the Commencement of Vitamin *D* Therapy. (Months.)	Number of Cases.
1	11
2	6
3	4
4	1
5	1

The fact that in a number of cases spontaneous cure occurred is important, for in ordinary mothercraft work this is undoubtedly the history of a high percentage of infants who develop rickets. The condition is undiagnosed; but as the child grows older it is likely to receive more sun exposure or its diet becomes modified, and as cow's milk replaces human milk, vitamin *D* is administered to most of these babies as a routine measure. Suffice it to say that in none of the cases of this series did the rickets persist beyond the first year of life.

Edith Robinson[39] showed that breast feeding with the addition of five drops of viosterol in oil was the most efficient method of preventing the development of rickets. This finding may not, at first, appear to harmonize with this investigation; however, Robinson took radiographs only every two months, and as the first visit of the mother to her clinic took place between the fifth and sixth week after the child's birth, it would have been possible for some of her subjects to develop rickets which had healed under the treatment before the next radiograph was taken. In her series only one baby out of 36 given breast milk and eight drops of viosterol developed rickets.

The short duration of the cases in which efficient treatment with a vitamin *D* preparation was given agrees with the findings of De Sanctis and Craig,[38] who found that cod liver oil at an estimated level of 380 to 460 international units per day prevented physical signs of rickets in 97% of the cases studied. Barnes, Brady and James[40] observed cure or prevention of rickets in all but two out of 64 babies receiving 840 international units of vitamin *D* per day.

I believe that in my investigation it has been possible by the use of regular radiographs at frequent intervals to keep an accurate check on the development and then the disappearance of the rhachitic changes, noting the effects of vitamin *D* therapy.

Summary.

The percentage absorption of calcium is probably the most important post-natal factor in both the development and the persistence of rickets during the first half of the first year of life.

Efficient administration of vitamin *D* to the infant in some form (at least 400 international units per day), commencing as early as practicable in the first month of life, will ensure that a high percentage of the infants will not develop rickets. Should rickets develop, the vitamin *D* brings about a cure within a few months in almost all cases.

The vitamin *D* produces these results by ensuring adequate absorption of calcium from the alimentary canal.

The Factors Responsible for the Development of Rickets in the Second Two-Thirds of the First Year of Life.—In this study six infants developed rickets in the second half of the first year of life. In two of the cases reported by Wake[34] the rickets made the first appearance after the fourth month. In both cases it was associated with diminishing calcium retention per kilogram of body weight. The late development of rickets is undoubtedly associated with the same factor responsible for the earlier persistence of rickets—namely, inadequate calcium absorption.

COMMENTS.

Throughout this discussion emphasis has been placed upon the importance of adequate amounts of vitamin *D* in the body of either a pregnant woman or an infant. It has been shown that this can best be achieved in most cases by the administration by mouth of some pharmaceutical preparation containing vitamin *D*. This is undoubtedly a provision wholly contrary to the natural process of child-bearing.

Primitive women obtain a maximum amount of exposure to the sun, as witness the New Guinea native. In an extensive experience in New Guinea I did not encounter one case of rickets. Women there chew large quantities of lime and their naked bodies obtain adequate exposure to the sun, leading to an adequate vitamin *D* supply within the body. Rickets is common amongst the Negroes in America, but they have assumed the white woman's clothes.

The women in Australia are extensively covered with clothes, especially in the winter months in the southern parts of Australia. At this time of the year the amount of ultra-violet light present in sunlight, even when this is available, is considerably less than during the summer. It would seem that the conventions of civilization have placed a heavy handicap upon modern women and that adequate supplies of vitamin *D* can, especially late in pregnancy, be obtained only by medication.

The two natural sources from which the young infant can obtain vitamin *D* are its mother or exposure to the sun. The average mother is hesitant to expose a young baby, particularly one under three months of age, to sunlight. This objection is even more real during the winter months. In these circumstances the young infant has to depend for its supplies of vitamin *D* solely upon the quantity provided by the mother in the milk, unless extra quantities are given by mouth.

Drummond *et alii*[41] investigated the amount of vitamin *D* in human milk and showed that the mean value of 26 samples was about six international units per 100 millilitres, but that the range was from two international units to eighteen international units.

Liu and his co-workers[37] demonstrated in their four cases that the administration of large doses of vitamin *D* to the mother greatly benefited the infant, bringing about a high retention of calcium from the maternal milk. It is quite possible that some of the vitamin *D* was passed on in the milk.

Grazer[42] found that 81% of infants born in the winter of 1939 in Frankfort had florid rickets, and inferred that the incidence was much greater than in those born in the summer months.

Investigations are required which will demonstrate that the vitamin *D* content of human milk can be altered by the administration of large doses of the vitamin to the mother. The little indirect evidence available suggests that this may be possible of achievement.

The accumulated evidence points to the fact that if the young infant is to receive optimal amounts of vitamin *D*, it will have to be provided directly, or through the mother by the administration of pharmaceutical preparations.

The Clinical Significance of Radiologically Evident Rickets.

So far we have been concerned with the number of infants who displayed radiographic evidence of rickets and have discussed possible causal factors with respect to both the origin and the subsequent duration of the rickets.

It should be once again emphasized that the changes seen in the bones fall into the category of mild rickets. From the point of view of practical pædiatrics it is important to know whether these rhachitic processes have (*a*) any lasting effect on the developing bones, (*b*) any influence on tooth development, (*c*) any effect upon the growth and development of the body as a whole.

The Effect of Rickets on the Developing Bones.

The experiments of Clark and Mrgudich[43] showed that even completely healed rhachitic bone (X-ray evidence) showed no return of the longitudinal orientation of the organic structures of the bone. Whether such bones are less efficient in the execution of the tasks of life has yet to be proved.

It has been held that rickets is an ætiological factor in certain bony deformities including knock-knees and bowlegs, and that certain changes in the shape of the chest wall (for example, Harrison's sulcus) are more common in infants who have had rickets than in normal infants. It is proposed to follow the infants in the present series for some years to study the relationship between the presence of rickets during the first year of age and the development of these bony changes.

The present information at my disposal is purely radiological, and reveals that three or four months after the rhachitic process has healed the bone has assumed a normal appearance. If the rhachitic process was severe, lines of arrested growth are evident; but with the passage of time even these fade.

The Effect of Rickets on Tooth Development.

The modern conception of rickets as a disease associated with defective calcium metabolism in general may bear an important relationship to dental caries. Massler and his colleagues[44] have shown that when the whole span of life is considered, the period of greatest susceptibility to hypoplastic defects in the enamel and the poorest calcification in the dentine lies between birth and ten months of age. This is the same period over which the rickets in the present series is spread. It has been shown that nearly 50% of all infants examined showed signs of defective bone calcification between birth and the age of one year. Massler *et alii* have shown that teeth record the same story of disturbed calcium metabolism at this age.

It is possible that the high incidence of dental caries may be associated with these disturbances of calcium metabolism. It is my intention to follow this series of subjects for as long as possible to study the incidence of dental caries.

The Effect of Rickets on the Growth and Development of the Body.

The rate of growth and the incidence of infections were studied in the rhachitic and normal children.

Growth.—In the infants who had radiographically proved rickets for one, two or three months no significant departure from normal rates of growth was detected. The weight chart of any rhachitic infant in this series could be duplicated many times from amongst the normal children. Those infants in whom the rickets had persisted for four or more months showed certain clinical features. They appeared to be heavier than they actually were, owing to the increased bulk of the soft tissues, which were less firm and more flabby than in a normal infant. This change would be in keeping with the low retention of calcium:phosphorus ratio, which is a feature of rickets. The relatively higher amount of phosphorus absorbed—owing to actual reduction in the calcium intake—is used for building soft tissues (Stearn[45]). After a duration of four or five months, rhachitic babies showed a retarded rate of growth; the weekly weight increments were not equal to normal. These infants appeared pale, suggesting an anæmia; but repeated hæmoglobin determinations failed to reveal any significant divergence from normal. Amongst the supposed classical signs of rickets epiphyseal enlargement was the most constant feature in the group of long duration. Hypertrophy of the costo-chondral joints was also common, but none of the other signs, including craniotabes, were observed in this series. The oldest infants in this series, who are now aged only two and a half years, were not old enough to show to any extent other bony changes, such as knock-knees, bowlegs and chest changes. The incidence of infections was no greater amongst the infants who had rickets than amongst the normal infants. It was my experience that it was impossible to diagnose on clinical grounds the presence of rickets in an infant when it had persisted for one, two or three months. If the condition lasts longer, then it is possible to recognize a higher percentage of the cases by the signs suggested above.

Summary.

Mild rickets of short duration seems to be without clinical significance at the time; but this in no way excludes the possibility that the defective calcium metabolism associated with the rhachitic process may produce lasting changes in the bones and teeth, which cannot be measured at that time. Suffice it to state that the presence of rickets means an abnormality in metabolism; it is certainly of a minor character, but it should not be permitted to occur if prevention is possible.

CONCLUSIONS.

1. The results of this investigation have shown that about 50% of infants developed mild rickets at some time during the first year of life. In a very high percentage of the cases studied the diagnosis could made only by radiographic examination of the growing end of a long bone, for in these cases the classical clinical features of rickets did not develop.

2. In view of the stigmata associated in the lay mind with the name of rickets, it is regrettable that these mild bony changes must be known by this name.

3. In at least 80% of the cases in which these rhachitic changes developed, cure was effected within a few months after the onset was recognized.

4. When the rickets persisted for more than three months some of the classical clinical signs appeared, particularly epiphyseal enlargement and hyperplasia of the costo-chondral junctions. The disturbed calcium metabolism appeared to have some effect upon the rate of growth, which was slowed down. An increase in the soft tissues, which became soft and flabby, was a characteristic of these cases.

5. The calcium intake of the mother, coupled with the adequate absorption of this calcium, appears to be the most important factor in the development of rickets early in the post-natal life of the infant. In the group in which the conditions for adequate calcium absorption by the mother prevailed, the percentage of infants who developed rickets was very much less than it was in the group in which the calcium absorption was inadequate.

6. Adequate calcium intake is associated with the consumption of at least a pint of milk per day, and the mother should take medicinal vitamin *D* to ensure adequate absorption of this calcium.

7. Post-natal feeding of the infant and even of the mother influences the duration and in some cases the development of rickets. It is not enough that the infant shall consume a certain quantity of human milk containing

the average amount of calcium; it is imperative that there shall be enough vitamin *D* in the body of the infant to ensure the highly efficient absorption of the calcium from the alimentary canal.

8. It is doubtful whether the requisite amount of vitamin *D* can be developed in the body of the infant by regular exposure to the sun, and it seems that the only satisfactory method of ensuring that the vitamin shall be available is its administration in some form by mouth. Limited research in Australia supported by much more extensive work overseas suggests that the optimal intake of vitamin *D* is about 400 international units per day.

9. It is possible that the desired amount of vitamin *D* can be passed on to the infant in the mother's milk, provided that the mother herself consumes moderately large doses of the vitamin.

RECOMMENDATIONS.

1. In order to reduce to a minimum the possibility of her infant's developing even mild rickets, every expectant woman should have a diet rich in calcium. This can be achieved only by the consumption of at least one pint of milk a day. This adequate intake of calcium should be accompanied by the consumption of medicinal vitamin *D* to ensure adequate absorption of the calcium. The increased vitamin intake alone without added calcium is of limited value.

2. When an obstetrician considers it necessary to withhold protein food from a pregnant woman and has included milk on his list of protein foods, he should provide calcium in some other form. If this is done, it should be realized that in some forms in which calcium is prescribed it is combined with a much larger molecule (for example, calcium lactate, calcium gluconate). An example of this is that about 120 grains (24 five-grain tablets or two level teaspoonfuls) of calcium lactate contain the equivalent amount of calcium to that present in one pint of milk.

The inclusion of milk in the group of protein foods is always a wonder to me. The average protein content of cow's milk is 3·5 grammes per 100 millilitres. A pint of milk would thus provide about 16 grammes of protein. The average daily consumption of bread—namely, 10·4 ounces—provides 25 grammes of protein, yet bread is not listed as a protein-rich food.

3. Every infant, including every breast-fed infant, should receive vitamin *D* by mouth as early as practicable in its post-natal life. It is desirable that this treatment should be commenced in hospital and continued during the child's attendance at the mothercraft centre.

It is doubtful whether enough cod liver oil emulsion can be given to young infants to provide the desired dose of vitamin *D* without the infant's digestion being upset. It is preferable to prescribe the vitamin *D* in one of the concentrated forms, of which one drop contains about 200 international units of vitamin *D*.

The optimal dosage of vitamin *D* is about 400 international units per day. Mothercraft nurses should be allowed to prescribe vitamin *D* in this concentrated form.

4. It is imperative that all premature infants and infants of low birth weight and twins should be given vitamin *D* in the concentrated form.

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Reports of Cases.

CONSERVATIVE TREATMENT OF MULTIPLE FIBROID TUMOURS: SUCCESSFUL PREGNANCY AFTER ENUCLEATION OF SIXTY-FOUR FIBROID TUMOURS OF THE UTERUS.

By CECIL CHARLES COGHLAN, M.B., F.R.C.S., F.R.C.P.I., F.R.A.C.S.,
Sydney.

THE case I am reporting here illustrates well the fact that, no matter how hopeless a case may seem with regard to ultimate fertility, the conservative treatment of fibroid tumours does sometimes well repay its adoption.

Clinical Record.

On May 10, 1940, Miss K.M., aged twenty-four years, of Greek parentage, came to consult me, complaining of hæmorrhage *per vaginam* and of a lump in her abdomen. She resided in Darwin and had consulted a practitioner there, who had told her that she had a tumour, that she needed an operation and that as a result of this she would never be able to bear children. As she was contemplating marriage in eight weeks she had come to Sydney for further surgical advice.

On examining her I noted that she was a well-developed girl of Mediterranean type; her blood pressure reading was 100 millimetres of mercury systolic and 70 diastolic; the hæmoglobin value was 75%. She had a hard, irregular mass rising from the pelvis, extending to the right of the abdomen as high as the umbilicus. Hæmorrhage was occurring at the time of the examination. The patient was virginal. A bimanual rectal examination disclosed that the tumour arose from the uterus. A diagnosis of multiple fibroids was made.

Considering her age and her expressed desire for children, I told her I would try to save the uterus for her, but advised postponement of her marriage for a while.

The patient entered hospital on May 14, 1940, and I enucleated 64 fibroid tumours from the uterus; these varied in size from two and a quarter inches in diameter to seedlings. "Avertin" and ether anæsthesia was used. At the conclusion of the operation, after sewing up the uterine wounds I stripped the peritoneum off the dorsum of the bladder and brought this over the fundus of the uterus so as to cover with peritoneum the whole uterine body.

Convalescence was uneventful, and examination on June 16 disclosed a uterus of normal size and freely mobile.

The next I heard of this patient was in a letter dated June 9, 1941, from a Brisbane colleague, who informed me that a Mrs. B., who had been Miss K.M., had consulted him and that she was two months pregnant. She had told him about the operation and wanted to know if it was safe to carry on. He asked me for details of the operation. On receiving the details he advised her to return to me for pre-natal care and confinement.

She came to Sydney and I saw her on November 19. Her pelvic measurements were slightly less than normal. The fundus was at the height of a thirty-two weeks' pregnancy. The position was vertex.

Considering the extensive scarring of the uterus and the small pelvic measurements, which would probably lead to a difficult labour, and also the great importance of this child, I eventually decided to perform a Cæsarean section, which I did on January 22, 1942, delivering the patient of a male child, weighing six pounds four ounces. I was very pleased that I had decided on a Cæsarean section, for the child had the cord twice round its neck and then under its left arm, and for good measure also had a true knot in the cord. I am certain that the child could not have survived a normal delivery. The uterus was well peritonealized; the scars of the previous operation were to be seen beneath the peritoneum, and there was only one filmy adhesion between the omentum and the lower end of the back of the uterus. No further fibroid tumours were seen.

To cover the wound in the uterus I used the same type of bladder flap as I had used before. This is very useful to extraperitonealize the scar in all Cæsarean operations.

Convalescence has been normal, and the patient is looking forward to a second child at a later date.

Comment.

I have considered this case worth recording for the following reasons: (i) the large number (64) of fibroid tumours enucleated; the number was doubly checked by the staff present at the operation; (ii) the successful use of the bladder flap of peritoneum to reperitonealize the uterus and thus avoid adhesions; (iii) the conception of a child reasonably soon after such an extensive operation, and in a type of case in which fertility is recognized as low; (iv) the happy ending.

Reviews.

WAR NEUROSES.

IN the hundred and ten compact and clearly written pages of his "Lectures on War Neuroses" the late T. A. Ross has packed the fruits of his psychiatric experience in two major wars.[1] To this is added his experience as director of the Cassel Hospital, and this was considerable. He was well known to many medical men as the author of "The Common Neuroses", a book which was the happy product of the marriage of common sense to simplicity and which gained for him a reputation for clear thinking and honest statement. The volume under review is stamped with these same commendable qualities. To its manifest lucidity it adds the charm of homely wisdom. It teaches without preaching, while to a large extent it disarms criticism by its candour.

The proper understanding of war neuroses is important because, unless properly diagnosed and handled, these neuroses can lead to years of misery and invalidism after the war, to say nothing of the cost to the community in pensions. "There are", as Ross writes, "men in England now leading aimless degraded lives because they drifted into this state during the last war." Ross pleads for a proper understanding of these neurotic conditions. He advocates treatment as early as possible. He believes that men with acute neurotic conditions should not be sent down the line, but should be treated in the forward area. Much, he claims, can be accomplished by persuasion and explanation, particularly by the doctor who is sure of his ground. He is emphatic that hysterical paralyses must be cured at a single sitting. Such patients are easily hypnotized; but Ross warns his readers of certain dangers inherent in such a procedure and suggests that this form of treatment should be left to those medical officers who have had some experience of it in civil life.

"The distinction between neurosis and cowardice is military and not medical." There is no means, Ross maintains, of judging from a psychiatric point of view whether a man is a coward who should be shot or a patient who should be treated. The malingerer in the army is the delinquent of civil life and his behaviour may be subconsciously motivated. We should know more about him before judging him.

Ross gives many examples of his commonsense psychotherapy. He exhorts medical officers to have patience and to deal truthfully with whatever symptoms are presented rather than resort to chicanery, however harmless it may appear. The prognosis in the acute neuroses, he asserts, is good if the treatment is prompt and the doctor sure-footed in his dealing with the case. It is otherwise when the neurosis has become chronic; and Ross does not think it is possible for the chronic patient ever to be cured sufficiently to resume active military duties, although he may successfully adapt himself to civil life.

This book is based essentially on personal experience. It is full of sound and practical advice unencumbered by theoretical speculations. It cannot fail to commend itself to the general practitioner who for the time being, engaged in military duties, may be thrown somewhat suddenly upon his own resources.

[1] "Lectures on War Neuroses", by T. A. Ross, M.D., F.R.C.P.; 1941. London: Edward Arnold and Company. Crown 8vo, pp. 123. Price: 6s. net.

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ACTIVATED STEROLS IN THE TREATMENT OF PARATHYREOID INSUFFICIENCY.

THE function of the parathyreoid glands is bound up with the metabolism of phosphorus and calcium. When the body is deprived of parathyreoid hormone the inorganic phosphorus content of the blood becomes increased and the calcium becomes less. Means points out that the rise of inorganic phosphorus in the blood is associated with a decreased output in the urine and is due, according to Albright, to this diminished excretion. Albright attributes the low output of calcium in the urine to a low level of calcium in the blood—in the face of an increased level of inorganic phosphorus in the blood, calcium is forced out of the blood into the bones. Normally the amount of calcium in the blood is in the neighbourhood of ten milligrammes per thousand cubic centimetres. In mild hypoparathyreoidism the level may be about eight milligrammes, and when the condition is severe, as low as four milligrammes. Parathyreoid insufficiency occurs in clinical medicine both as post-operative tetany following surgical operation and as spontaneous hypoparathyreoidism, a condition analogous to myxœdema. Among the measures adopted to combat the parathyreoid insufficiency is the administration of activated sterols. These are complex substances and a good deal of uncertainty exists as to the place they should occupy in treatment. In the circumstances it seems advisable to place before readers of this journal the salient features of a review by F. C. McLean, of the Department of Physiology in the University of Chicago, on the activated sterols in the treatment of parathyreoid insufficiency.[1]

The management of hypoparathyreoid conditions with activated sterols is of special importance because substitution therapy by the administration of a solution of parathyreoid substance is limited to more or less acute conditions in which treatment needs to be continued for days or weeks rather than for months. Activated sterols have two advantages—the body does not develop a tolerance to them and they may be given, indeed preferably are given, by mouth. Their action, however, is somewhat delayed, so that for the relief of acute parathyreoid tetany a solution of parathyreoid gland may be indispensable. If ergosterol is irradiated, a chain of reactions takes place. Among the substances produced by such irradiation are tachysterol, toxisterol and calciferol. McLean draws attention to the work of Holtz and Schreiber, who in 1930 distinguished between the antirhachitic action of the irradiation products of ergosterol and those actions which are responsible for the toxicity of certain of these products. Holtz and Schreiber were unable to make a preparation which was antirhachitic but non-toxic; they were, however, able to destroy most of the antirhachitic action and at the same time to retain and actually to increase the toxic effects—hypercalcæmia and pathological calcification in the soft tissues. The principle responsible for these effects they called the *Calcinosefaktor*. Holtz and Schreiber were responsible for the further study of this factor, which McLean calls the "calcæmic principle", and eventually for its introduction into clinical use in the form of dihydrotachysterol, originally known and often referred to as "A.T.10" (*Antitetanisches Präparat Nr.* 10). The action of the "calcæmic principle", McLean points out, is non-specific and is shared by a number of substances, of which only vitamin D_2 in the forms of crystalline calciferol and of viosterol, and dihydrotachysterol have received sufficient investigation and clinical trial to warrant a review of their usefulness in the treatment of hypoparathyreoidism. It should be pointed out that viosterol is the non-proprietary name adopted by the Council on Pharmacy and Chemistry of the American Medical Association for all "acceptable" preparations of irradiated ergosterol. It should also be mentioned that dihydrotachysterol differs from calciferol only in the saturation of a double bond, but that this difference is sufficient for the disappearance of all but a small fraction of the antirhachitic action and for a decided enhancement of the toxicity. Dihydrotachysterol is formed when tachysterol is treated with sodium and propyl alcohol. McLean does not consider the use of vitamin *D* for other purposes than to regulate the concentration of calcium in the plasma. He finds in the literature an unwarranted implication that the beneficial effects of dihydrotachysterol are specific for that substance; he presents evidence to the contrary. He concludes that similar, if not identical, effects may be obtained with several of the activation products of ergosterol and cholesterol and that there is no satisfactory evidence that the therapeutic effects of dihydrotachysterol are superior to those of vitamin *D* when the latter is given in comparable doses. Though in stating this we are anticipating McLean's conclusions, it is convenient because it brings us to the question of dosage. Confusion has arisen over the use of vitamin *D* in the management of hypoparathyreoidism because there are for this substance two distinct dosage ranges. The prophylactic and curative range for the antirhachitic effect is ordinarily from 700 to 1,000 international units a day; the dosage needed to raise a subnormal serum calcium level is anything from 60,000 to 200,000 international units a day and may even be higher over short periods. Adminis-

[1] *The Journal of the American Medical Association*, August 23, 1941.

tration of dihydrotachysterol has been completely divorced from the system of units by which vitamin *D* preparations are standardized; it is standardized only in terms of its "calcæmic principle" and is thus free from the confusion that surrounds the administration of vitamin *D*. The "calcæmic principle" is common to both vitamin *D* and dihydrotachysterol and is responsible for the manifestations of overdosage. The effectiveness of the "calcæmic principle" is influenced by the degree of parathyreoid insufficiency, by the intake of calcium and phosphorus, by the female hormone and perhaps by other variable physiological factors. For this reason there are no absolute standards of dosage for the production of therapeutic or toxic effects. McLean deals with the dosage question in two sections, initial dosage and maintenance dosage. Relatively large doses of dihydrotachysterol have been used and recommended for the initiation of treatment in acute attacks of hypoparathyreoidism. In chronic parathyreoid insufficiency it is more common to initiate treatment with one to four cubic centimetres of the oily solution every day (five to twenty milligrammes of the basic substance). At least ten milligrammes of calciferol are required for a calcæmic action equivalent to that of five milligrammes of the basic substance; therefore a corresponding daily dose of calciferol would be ten to forty milligrammes or 400,000 to 1,600,000 international units of vitamin *D*. McLean points out that while these doses have the appearance of being enormous, owing to the system of units employed, there is little evidence of their harmful effects when they are administered over relatively short periods. He also holds that much of the earlier literature on the toxicity of vitamin *D* preparations must be discarded, owing to a wide use of a preparation of irradiated ergosterol with a low vitamin *D* and a high toxisterol content. (Toxisterol, McLean remarks, has been stated by Shohl to have toxic actions qualitatively different from those of the other substances that have been mentioned.) In the literature of the past few years he has been able to find no report of serious toxic effects from large initial doses of dihydrotachysterol and only one instance in which serious results followed large doses of vitamin *D* taken over a relatively short period of time. This was a case in which a medical practitioner died after having taken by mistake 2,300,000 international units of vitamin *D* every day for eighteen days. In regard to maintenance doses, McLean has found no record of fatal poisoning after the administration of dihydrotachysterol. He mentions three deaths among infants after the administration of vitamin *D*, but these are among the records of many observers. Those who did record untoward effects stated that the patients recovered. McLean has found little evidence in the literature of chronic injury in man resulting from the administration of dihydrotachysterol or of products of vitamin *D*, but he adds that the possibility of such injury is by no means excluded. Reed, Struck and Steck have stated that their extensive use of vitamin *D* in large doses has not indicated that any chronic injury is to be expected if toxicity has not occurred, and that even when toxic effects have been observed it seems improbable that any residual chronic effects will follow. This, McLean points out, is in agreement with a recent report by Holtz on the prolonged use of dihydrotachysterol. McLean discusses the mode of action of activated sterols in the light of the work of many observers, including himself. He concludes from comparative studies that there is a qualitative difference between the actions of toxisterol on the one hand and vitamin *D* and dihydrotachysterol on the other. The studies discussed by him, however, fail to disclose any difference in the actions of the two last-mentioned substances on which a preference for either one or the other may be based. He thinks that further carefully controlled comparative studies, in both clinical and experimental hypoparathyreoidism, are required before it will be possible to say finally that calciferol and dihydrotachysterol are of equal value in the management of the condition or that either has advantages over the other. McLean also discusses clinical experiences with both dihydrotachysterol and vitamin *D*. For details of this part of the review readers are referred to the original document. It must suffice to state that there is needed a larger volume of experience with vitamin *D*, comparable with that already accumulated for dihydrotachysterol. From the practical point of view of the clinician the chief conclusion is that the toxic effects of the substances mentioned, while real, have been over-emphasized. The substances can be used, but must be used with care and with proper precautions—"the immediate dangers are apparently no greater than in the case of insulin". Frequent determination of serum calcium must be made, not only during the initial stages of treatment, but also after maintenance doses have been determined. McLean thinks that the Sulkowitch test (described by F. Albright in *The Journal of the American Medical Association* of June 24, 1939, at page 2592), by which the excretion of calcium into the urine is observed by a simple and roughly quantitative method, may serve to avoid the necessity for frequent examinations of the blood. As a final plea McLean claims that the term "hypervitaminosis *D*" should be abandoned because it "is misleading and non-descriptive and is a deterrent to the legitimate and desirable use of vitamin *D* products in the treatment of hypocalcæmia".

McLean's review is important. He writes as one who knows his subject (his own contributions to the literature should convince any who doubted this statement) and his review, which appears as a "special article", is published under the auspices of the Council on Pharmacy and Chemistry of the American Medical Association. The treatment of parathyreoid insufficiency with activated sterols is a subject of absorbing interest, for it must be clear that biological chemists have still much to discover about sterols and allied substances. The response of the parathyreoid to treatment with these bodies is part of the larger and but partially explored field of endocrinology. This is a study which no practitioner of medicine, whatever his particular interest, can afford to neglect.

Current Comment.

THE TREATMENT OF HÆMATEMESIS.

THE treatment of peptic ulcers and especially ulcers associated with hæmorrhage has for many years provided a constant subject for discussion between physicians and surgeons with the periodical ascendancy of one or other school of thought. In spite of all the investigations and discussions, this subject is still in a state of flux and is likely to remain so until comprehensive surveys are carried

out in the larger clinics of the world, and even when this has been done the question may not be settled. At present most physicians and surgeons have to rely on clinical impressions derived from relatively small series of cases which do not stand statistical investigation, and from which erroneous conclusions may be drawn by the unwary.

In any discussion of the treatment of hæmatemesis, hæmorrhage from lesions such as œsophageal varices, which do not lend themselves to operative interference, may be omitted. It is important to differentiate between hæmorrhage from gastric and duodenal ulcers, and to divide the patients into groups dependent on their age and on their previous history. In the history, special reference should be paid to the distinction between small repeated hæmatemeses and massive isolated hæmatemeses, and to the length of time elapsing between the onset of the bleeding and the commencement of treatment. It is only by such a subdivision of cases that clarity is attainable in conclusions drawn from them. In concealed hæmorrhages there is the difficulty of determining how much blood has been lost, and a rough estimate must be made from the rapidity of the pulse, the low blood pressure, the symptoms of shock; but it must be remembered that the use of such rough estimates for the purposes of classification is open to many fallacies.

Recently Waltman Walters and W. H. Cleveland,[1] of the Mayo Clinic, have adopted such a classification of their cases and have reported their operative results. These authors pointed out that with rest, blood transfusion, and proper diet, cessation of the bleeding occurs in most cases of acute massive hæmorrhage from gastric or duodenal ulcers when the patients are aged less than 45, and in about 70% of cases when the patients are aged more than 45. Obviously, if this is so, the results from conservative measures cannot be greatly improved by surgical intervention in the younger age group. It is in the older group that a reduction of mortality is possible, but part of the difficulty is how to recognize the 70% of patients in this group who will recover from the effects of the massive hæmorrhage under medical treatment alone and whose lives might be endangered by operation. There is also a difficulty in recognizing which of the remaining 30% would be saved by operation. These criteria, as mentioned above, will be elicited only by the study of large series of cases. The importance of the time factor in the cases of massive hæmorrhage in which operation is performed has been repeatedly stressed by Finsterer, of Vienna. Finsterer divided his patients with bleeding peptic ulcers into two groups—those operated on within forty-eight hours of the onset of a massive hæmorrhage with a 5·1% mortality, and those operated on later than forty-eight hours from the onset of a massive hæmorrhage with a 29·7% mortality. Finsterer's report comprises the results from 152 cases and is certainly impressive. As Walters and Cleveland point out, the great difference between the emergency operations performed at the time of or shortly after the onset of the hæmorrhage and those performed several days later is dependent on the nephrotic condition, with among the latter group retention of nitrogen and frequently œdema which greatly increases the operative risk. Walters and Cleveland's series of cases of massive hæmorrhage was smaller than Finsterer's, but similar results were obtained in the two series. The presence of arteriosclerosis and the difficulty of constriction and thrombosis in an eroded thickened artery are the probable explanations for the continuation of the bleeding and the higher mortality rate among the older age groups. In the younger patients repeated massive hæmorrhages rarely occur, but whatever the age of the patients, they may be indications for operation. With this exception massive hæmorrhage from peptic ulcers in the younger patients should be treated conservatively. In patients over the age of forty-five with a massive hæmorrhage immediate operation may be urged. Finsterer's experience emphasizes the seriousness of a few days' delay in the treatment of these cases. If operation is not possible within forty-eight hours of the onset of the hæmorrhage it should be delayed until the patient has completely recovered from the effects of the hæmorrhage. The type of operation required in cases of peptic ulcer with massive hæmorrhages depends on the situation of the ulcer and any adherence to neighbouring structures.

For the diagnosis and treatment of massive gastric hæmorrhage John V. Bohrer[2] inserts a small tube such as a Levine's into the stomach through a nostril, aspirates the liquid blood and washes the stomach with saline until it is free from blood clots. The removal of the blood from the stomach is said to prevent nausea and vomiting. If after the stomach is entirely empty, bright red blood continues to emerge from the tube, it is apparent that hæmorrhage is continuing. Bohrer regards this as a definite indication for immediate operation; but in a young patient with hæmorrhage from an acute gastric ulcer it is difficult to believe that this treatment would not tend to perpetuate the bleeding, which, with more gentle and conservative treatment, would probably cease. Bohrer partly answers this objection by the statement that if the clot is so easily removed it must be friable and useless, and is therefore better removed. Following the work of Meulengracht, the old idea of starvation for bleeding peptic ulcers is gradually but slowly being abandoned. A more acceptable diet is one that will absorb acid and prevent the action of the digestive juices on the ulcer and especially on the clot in the eroded vessels. The diet must also contain vitamins so that, if it is persisted in, there will not occur a recurrence of gastric hæmorrhage due to a mild and not obvious scurvy.

Just as there is a wide divergence of opinion between some physicians and surgeons as to the indications for operative interference in cases of hæmatemesis, so also do many physicians differ from their colleagues as to the indications for transfusions for these patients. The objection raised by a small minority is that transfusion, by raising the blood pressure, may dislodge the clot from the bleeding vessel with subsequent recurrence of the hæmorrhage. No proof of this theoretical objection has ever been produced. However, to continue the theoretical argument, such a risk would be minimized by the slow, continuous drip method of transfusion. On the other hand a severe anæmia may result in a lowering of resistance to infection, in the production of an extrarenal azotæmia due to the low blood pressure with a resulting decrease in the urea clearance and an increase in the blood urea, and in anoxæmia due to poor absorption and transportation of oxygen, which may cause necrosis of vital tissues, not only in elderly people with sclerosed vessels, but also in young people with normal vessels if the anæmia is allowed to persist.

The treatment of chronic hæmorrhage from a peptic ulcer is quite a different and distinct problem from the treatment of massive hæmorrhage, and most clinicians will agree that it is in chronic hæmorrhage, unless the ulcer symptoms become intractable, that medical treatment is indicated. Walters and Cleveland recommend partial gastrectomy for those patients with peptic ulcer and chronic hæmorrhages who have failed to respond to medical treatment. In the case in which the ulcer was on the posterior wall of the duodenum (the most frequent site for a bleeding duodenal ulcer) and could not be removed at operation, Walters and Cleveland obtained satisfactory results in 94% of cases by oversewing the ulcer, ligating the vessels, and performing a partial gastrectomy. By "satisfactory" is meant that there has been no further hæmorrhage and no symptom of ulcer. This is in distinct contrast to the 24% of bleeding duodenal ulcers which continued to bleed following gastrojejunostomy performed at the Mayo Clinic in the previous five years.

If, because of the age of the patient or the duration of the history, the position of the ulcer, the size of the ulcer or the results of gastric analysis, malignant disease is to be suspected, A. W. Allen and C. E. Welch[3] hold that surgery is indicated; in fact they think that opera-

[1] *Annals of Surgery*, October, 1941.
[2] *Ibidem.*

[3] *Ibidem.*

tion should be undertaken if any of these criteria are present. These authors also recommend operation in younger patients with gastric ulcers less than one centimetre in diameter if healing is not complete within one month of the start of medical treatment; for, as they state, the risk of surgery is less than the risk of leaving a malignant ulcer *in situ*.

During the past ten years Walters and Cleveland have been able to report a reduction in the operative mortality for all cases of bleeding peptic ulcer from 10·3% to 4·1%. They attribute this decrease in operative risk to improvement in pre-operative and post-operative care, to the use of chemotherapy, to a better understanding of vitamin deficiencies, to more rigid indications for partial gastrectomy, to improvement in the treatment and the prophylaxis of post-operative pulmonary complications, and to the elimination of operative procedures which have proved of least merit.

The three papers that have been mentioned must be regarded almost as *ex parte* statements or at least as tinged with surgical enthusiasm. We do not doubt that the authors of these papers obtain the results that they claim. Such results, however, are for the highly expert surgeon. Most practitioners will hesitate before they submit to operation a patient who has had a large hæmorrhage from the stomach; and many will insist (and rightly) on a fair trial of medical treatment before a decision to operate is made, even in the treatment of a patient with a chronic ulcer. We realize that an effort to cure a patient by removal of tissue is a confession of failure; so, too, for that matter is medical treatment of ulcer, for if our knowledge was complete we would know the cause of ulcer and would be in a position to prevent it. One certain statement can be made in conclusion—if surgical treatment of any kind appears likely to be called for, delay must not be so long that the patient is *in extremis* before the surgeon sees him.

THE PROPHYLAXIS OF PULMONARY EMBOLISM.

THE coagulation of blood within the veins presents a complex problem. In some instances it is benign in character and results. Of this nature is the intravascular clotting which occurs inside a divided vein and forms part of the process which arrests hæmorrhage. Also beneficial is the obliteration of varicose veins by clot which later organizes. In this instance admittedly a further factor is involved, namely, the reversed flow of blood which occurs in varicose veins, and which is stopped by their closure. Turning now to some of the less favourable examples, and confining our attention, for present purposes, to the lower limbs and pelvis, we find that thrombosis most commonly follows operation, childbirth or illness and may lead to local or remote effects. The local effects include pain and swelling in the extremity. The remote effects are embolic and follow the impaction in the pulmonary arteries or their branches of portions of clot which have become detached. When the embolus is large the result is usually fatal, and the event tragic. A patient of or past middle age, who has been making an apparently uneventful recovery from an operation, succumbs in a few minutes. Treatment of the established thrombosis or of the remote embolism has hitherto been unsatisfactory. The minor instances of each subside more or less completely with or without treatment (unless further complications supervene), but the major examples demand an improvement in our methods. Gross œdema of a lower limb following a femoral thrombosis often requires for its control lifelong dependence on the support of an elastic stocking, while anyone with experience of Trendelenburg's operation of pulmonary embolectomy will appreciate that for clinical, practical and technical reasons it is of extremely limited use. In this problem, therefore, even more than in most, attack along preventive lines is indicated. Certain aspects of preventive treatment have been appreciated and employed for many years, though there is reason to doubt whether they have been carried out as widely or as thoroughly as is to be desired. Among these are gentle handling of tissue, measures to prevent or to combat infection, the avoidance of dehydration, and the early institution of exercises to overcome venous stagnation.

These and other prophylactic measures have been picturesquely presented and reviewed by Hugh Robertson.[1] An agent which has become available recently is heparin, or "anti-prothrombin". C. Crafoord and E. Jorpes[2] have shown that regular treatment with heparin is an almost certain preventive of post-operative thrombosis. Unfortunately the routine treatment of all patients with heparin after operation is impracticable on account of the cost, time and effort involved. Gunnar Bauer[3] stresses the value of heparin, and considers that advantage may be taken of its properties by the selection of the cases in which it is to be administered. Bauer points out that while heparin has no effect on established thrombosis, it will prevent its extension. He has directed his effort towards the detection of patients who are "just getting thrombosis". The early and transitory stage of thrombosis evades recognition by ordinary clinical methods, but by means of phlebography, thrombotic conditions in an early stage may be revealed. Twenty cubic centimetres of "Perabrodil" are injected slowly into a vein behind the outer malleolus, and an X-ray picture is taken of the lower extremity. In the normal limb the deep veins fill through communicating veins, and show up clearly on the film. If the deep veins are clogged by thrombotic masses, then filling defects or a complete absence of the contrast shadows will result. Phlebography confirms the view that thrombosis commences in the deep venous trunks in the lower part of the leg. From here the process extends proximally, until a long eel-like clot is waving freely in the blood stream. When this stage has been reached either of two results may occur. If the thrombus breaks at any point the proximal portion will be carried towards the heart and will form an embolus. If it does not break, it will eventually become adherent to the vessel wall. In this case it will lead to periphlebitis and the consequent blocking of perivenous lymphatics. The result of the combined venous and lymphatic obstruction will be gross œdema of the extremity, the *phlegmasia alba dolens* of the puerperium being an example. To secure a timely and at the same time reliable diagnosis of early thrombosis by phlebographic examination, it is necessary to be watchful for the case in which the examination is to be made. Heed must be given to such diagnostic "straws" as the slightest unexplained rise in the pulse rate or the temperature, the faintest signs of trouble in the legs such as a transitory aching, or the least suggestion of firmness or tenderness in the calf. The application of phlebography will enable treatment to be instituted for patients showing evidence of thrombosis, while a negative result will avoid the inclusion of such a patient among the "successful" results of any particular form of treatment. Bauer presents a series of thirty-two patients in whom thrombosis in the deep calf veins was diagnosed by phlebography, but who did not receive treatment with heparin. In only eight did the condition remain local. In the remaining 24 the thrombosis spread to the femoral vein and pronounced phlegmasia resulted. Two patients died, one from pulmonary embolism and the other from thrombosis of the superior mesenteric vein. Since 1940 Bauer has used heparin in the abortive treatment of thrombosis. The number of cases observed has been small (21), but the results have been uniformly good. Temperature fell and pulse rate subsided, and swelling, aching and tenderness in the leg vanished. In no case did thrombosis spread, and no patient suffered from pulmonary infarct. Heparin was administered for three to five days, the usual dosage being 100 milligrammes given thrice daily by the intravenous route. J. Fine and J. B. Sears[4] have written recently on the prophylaxis of pulmonary embolism. The

[1] *The American Journal of Surgery*, Volume XLI, 1938, page 3.
[2] *The Journal of the American Medical Association*, Volume CXVI, 1941, page 2331.
[3] *Archives of Surgery*, September, 1941.
[4] *Annals of Surgery*, November, 1941.

method they employ is the division between ligatures of the femoral vein at a point just distal to its junction with the *profunda femoris* vein. Although these workers claim that this procedure is without serious untoward effect and has a definite preventive value, it is clearly practicable only in selected cases. The selection of these cases is made by clinical and phlebographic methods similar to those discussed above. The operation of ligature of the femoral vein, despite the enthusiasm of its protagonists, cannot be regarded as a step lightly to be embarked upon after surgical operation, and would appear, even in the selected cases in which embolism is impending, to be indicated only when heparin cannot be obtained. In the strictest sense, however, neither of the two recent methods is entirely prophylactic. With each a semi-defeatist attitude is taken of waiting for thrombosis to commence, and then of attempting to limit its extension. At the moment heparin and femoral vein ligation, used in conjunction with phlebography, probably deserve careful consideration in relation to the basic problem, and it is regrettable that in this country shortage of time and materials makes confirmatory work difficult at present. Reliance must be placed mainly, therefore, on those older measures which were recounted earlier in this commentary, and which for the sake of emphasis may be repeated. They comprise early and continued post-operative exercises, active or passive as necessary, gentle surgery, avoidance or treatment of infection, and preparation of the patient to combat dehydration and malnutrition.

SULPHANILYLGUANIDINE.

SULPHANILYLGUANIDINE was introduced by Roblin in 1940. Although this drug was shown to have a relatively high solubility in water (190 milligrammes per 100 millilitres at 37° C.), it was found to be poorly absorbed from the intestinal tract; and it was this property that has suggested the use of this drug in the treatment of bacillary dysentry, ulcerative colitis and other infections of the alimentary tract. Other sulphonamides such as sulphapyridine and sulphathiazole are more efficient antibacterial agents than sulphanilylguanidine, but the former two drugs, being rapidly absorbed from the bowel, cannot greatly influence the intestinal organisms; and as was therefore to be expected, neither sulphapyridine nor sulphathiazole was found to be efficient in the treatment of bowel infections. To date, the greatest use for sulphanilylguanidine has been for the treatment of bacillary dysentry, in which disease encouraging results have been obtained. Recently Warfield M. Firor and Edgar J. Poth[1] suggested the use of sulphanilylguanidine for the pre-operative preparation of patients requiring operations on the colon. These authors found, however, that patients on the same diet and on the same dosage of the drug (usually 50 milligrammes per kilogram of body weight every four hours) showed great variation in the effect produced by the drug on the numbers of the intestinal organisms. Patients with an ulcerating carcinoma of the colon showed little or no fall in the number of bacteria in the stool. These authors investigated the intestinal flora of only a small number (20) of cases of ulcerating carcinomata of the colon from this aspect. If it is subsequently proven that an ulcerative malignant lesion of the bowel neutralizes the bacteriostatic activity of sulphanilylguanidine, the use of this drug in surgery will necessarily be limited to those cases in which the ulcerative malignant lesion has been previously removed by a Paul-Mikulicz or related procedure, or in which the operation is being performed for some condition other than an ulcerative malignant lesion. Firor and Poth suggest that the inhibiting action of these lesions is probably explained by their production of an anti-sulphonamide factor, similar to that present in other purulent exudates, which rapidly counteracts the anti-bacteriostatic action of any of the sulphonamides and renders them ineffective.

[1] *Annals of Surgery*, October, 1941.

While the use of sulphanilylguanidine before and after operations on the intestines is still in the experimental stage, the drug is non-toxic, and if available, there seems to be no contraindication to its wider trial in such cases. Such a suggestion as that of Firor and Poth will be important if it points the way to the development of this or other sulphonamides capable of considerably decreasing the intestinal flora, and thus making surgery of the large bowel safer than it is at present.

HALITOSIS.

THERE seems to be a general impression that persons suffering from halitosis should be treated by dental surgeons or by medical men specializing in diseases of the nose, throat and oral cavity. Everyone will agree that decaying teeth or suppurative conditions of the gums and also certain lesions of the nose and throat will make the breath foul. But, at least in Australia, oral hygiene has in recent years been pursued much more ardently than of yore, and attention is paid to the nose and throat. We may therefore agree with B. B. Crohn and R. Drosd when they state[1] that the teeth, the gums and the pharynx account for true fetor *ex ore*, but do not explain the malady known as halitosis. These observers have made an ingenious investigation and have been impelled to do so by contradictory observations previously reported. In 1935 H. W. Haggard and L. A. Greenberg reported that the odour of garlic on the breath resulted only from particles retained in the crevices of the teeth. They claimed to eliminate the odour from the mouth by the use of chlorinated washes and other preparations. Later, in 1936, M. H. Blankenhorn and C. E. Richards opposed the view of Haggard and Greenberg. They inserted garlic by a gastrostomy tube into the stomach of a patient who had complete stenosis of the œsophagus, and were able to recover the odour of garlic on the breath. Crohn and Drosd seized an unusual opportunity to study this subject. They had a patient, seriously ill with ulcerative colitis, who had undergone colostomy for obstruction low in the intestine, and also ileostomy. In the ileostomy operation the terminal portion of the ileum had been divided and both stomas were anchored in the abdominal wound. In addition the patient had a "toxic psychosis", and an indwelling Levin gastric tube had been inserted and was maintained in place for feeding. Conditions could not have been more favourable to experiments with garlic. The experiments undertaken included administration of garlic through a gastric catheter, administration of garlic into the colon and the distal segment of the ileum, and administration into the proximal segment of the ileum. The stomach plays no part in absorption of garlic, some absorption takes place from the colon and distal part of the ileum, but absorption is mainly from the upper part of the small intestine. At this point it is interesting to note an observation by Goodman and Bearg, that garlic is and can be carried by the blood stream—they demonstrated on the breath of new-born infants the odour of garlic which had been ingested by the mother before delivery. Crohn and Drosd also made a series of observations on patients with carcinoma of the pancreas and complete obstruction of the bile passages in whom biliary drainage had been established by surgical means. Garlic is stored in the liver. "Odour in the bile and on the breath appeared almost simultaneously, but that on the breath always persisted for one or two hours after the bile had ceased to be malodorous." The question naturally follows: "Can anything cover the odour of garlic?" Crohn and Drosd have examined other substances and find that peppermint is excreted on the breath when given in moderate doses. It would, as they state, be alluring to think of covering a garlic odour with peppermint. The point is well worthy of investigation.

[1] *The Journal of the American Medical Association*, December 27, 1941.

Abstracts from Medical Literature.

RADIOLOGY.

The Appearance and Nature of Cyst-Like Areas in the Distal Femoral Metaphyses of Children.

LESTER WARREN SONTAG AND S. IDELL PYLE (*American Journal of Roentgenology*, August, 1941) state that translucent cyst-like areas, usually of one to three square centimetres in size, are frequently found in the medial aspect of the distal metaphysis of the femur in skiagrams of children between the ages of two and nine years. These circumscribed areas are the result of local removal of bone salts. They occurred at one time or another in 53% of the authors' series of boys and in 22% of the girls. Their average duration was twenty-nine months. They are asymptomatic and disappear spontaneously. They are frequently bilateral. The condition may be due to the inclusion of small groups of cartilage cells in the invading metaphysis, which stimulates the activity of osteoclasts. The resultant bone destruction permits growth of the cartilage cells. These pseudocysts may not be greatly different from solitary bone cysts except in the degree to which they progress. This benign condition must be differentiated from such infections as Brodie's abscess, syphilitic abscess and pyogenic abscess.

Deformities and Fractures of the Vertebræ as a Result of Senile and Presenile Osteoporosis.

LOUIS NATHANSON AND ALEXANDER LEWITAN (*American Journal of Roentgenology*, August, 1941) present a series of cases in which deformities, compression and fractures of one or more vertebræ resulted from senile and presenile osteoporosis. In none of these cases could the pathological change in the spine be accounted for by a specific disease entity. The vertebral lesion was discovered in each instance as an incidental finding to some other radiological investigation. No pain or very little pain was referred to the spine. Vertebral collapse associated with osteoporosis occurs fairly frequently in senile and debilitated states and should be differentiated from cancer and other specific diseases. The diagnosis of vertebral collapse *et cetera* due to senile and presenile osteoporosis as a rule is made by elimination. The absence of pain and the presence of characteristic deformities of other vertebræ are significant.

Mediastinal and Pulmonary Changes in Erythema Nodosum.

L. W. PAUL AND E. A. POHLE (*Radiology*, August, 1941) review the theories as to the ætiology of *erythema nodosum* and stress the importance of considering tuberculosis as a causative factor. They state that in a given case of mediastinal and hilum enlargement the presence of a coexisting or recent *erythema nodosum* should be of great value in establishing the nature of the adenopathy, since, even in adults, such a combination is most likely to be of tuberculous ætiology. Conversely, when radiological examination of the chest of a patient with *erythema nodosum* reveals hilum adenopathy, the tuberculous ætiology of the former can be surmised. It is suggested, therefore, that all patients with *erythema nodosum* be subjected to radiological examination of the chest as part of the diagnostic study. Even where this reveals no pathological change, follow-up examinations should be made before the patient is considered to be free from tuberculosis.

Röntgen Findings in Alkaptonuric Ochronosis.

M. POMERANZ, L. FRIEDMAN AND I. TUNICK (*Radiology*, September, 1941) report two cases of alkaptonuric ochronosis showing the triad alkaptonuria, ochronosis and arthritis. The disease represents a clinical entity which has its genesis in a rare metabolic anomaly. Skiagrams of a typical case display extensive calcification of the intervertebral disks with ankylosis of the spine. Similar calcifications occur in tendon sheaths, bursal sacs and in synovial membranes.

Tuberculosis of Bronchial Lymph Glands.

NILS WESTERMARK (*Acta Radiologica*, Volume XXI, Fasciculi 4 and 5, 1940) states that very useful information of the hilum is obtained if, in addition to the taking of radiograms in the ordinary way during inspiration, pictures are taken while the patient is carrying out Valsalva's experiment. As is well known, the vascular design is diminished in the lungs as well as in the hilum during such experiments. The heart and the large vessels are also considerably reduced in size after this experiment. On the other hand, it has no effect on the lymph glands or other structures in the hilum and mediastinum. Smaller lymph glands, which earlier could be distinguished in the skiagram because of their being obscured by the large vessels, may become visible during this experiment. In such cases, when in the interpretation of a shadow in the skiagram it may be doubtful whether it is caused by a vessel or some lymph gland, a Valsalva's experiment should always be carried out. If the shadow is caused by a lymph gland or some tumorous formation, it appears unaltered after the experiment; should, on the other hand, the shadow be caused by some vessel, it diminishes in size or disappears altogether at the experiment. In every case in which tuberculosis of the lymphatic glands of the hilum is suspected and the glands cannot be directly observed in the skiagram, a Valsalva's experiment should be carried out. If the radiologist has been able to verify the presence of enlarged lymph glands, it is then a big problem to decide whether this enlargement is the result of tuberculous changes or of some other pathological condition in the gland. A bronchopneumonia even if slight extent may cause a more or less pronounced hilar lymphadenitis. In these cases too the lymphadenitis is regional to the pulmonary field. A hilar lymphadenitis in connexion with bronchopneumonia is rarely, however, of the same extent and density as tuberculous lymphadenitis. At the first examination it may be impossible, however, to make a definite radiological differential diagnosis. The bronchopneumonic changes as well as the consequent hilar lymphadenitis run a very acute course, the changes generally disappearing fairly soon or diminishing considerably. On the other hand, it is found that a primary tuberculous focus with consequent hilar lymphadenitis runs a more chronic and often progressive course. A tuberculous primary focus and tuberculosis of hilar lymph glands clear up very much more slowly than bronchopneumonia with secondary lymphadenitis.

Changes in Bones, Joints and Soft Tissues Associated with Disease or Injury of the Central Nervous System.

JAMES F. BRAILSFORD (*British Journal of Radiology*, October, 1941) states that besides the arthropathies described by Charcot, found in association with *tabes dorsalis*, syringomyelia, *spina bifida*, myelitis, post-encephalitis, paraplegia, injuries to the posterior nerve roots and other affections of the central nervous system, certain other changes, usually of much less importance, may be seen to follow injury to the central nervous system, notably the so-called localized *myositis ossificans* and ectopic bone formation. In a small percentage of patients, at the site of contusions, more commonly in the region of the femoral shaft and lateral aspect of the ilium, a sequence of changes, recognizable on radiographs, occur which bear a close resemblance to those seen in the soft tissues around a neuropathic joint. So close is the resemblance that one is forced to believe that some common factor is in operation in both, that is, a lesion of the central nervous system. In this condition the patient may complain of some limitation of the movement of a limb and discomfort over a hard swelling of the part, the skin of which may be reddened and tender. If recent, some sign of discoloration of the skin may be seen, but often the patient cannot remember having received any injury to the part. Radiographs of the part will furnish a clue to the nature of the lesion. The earliest recognizable feature, which is not visible for three to four weeks after the trauma, is the deposition of flocculent calcium near the bone in the region of the tumour. As the amount of calcium deposited in the soft tissues increases during the following month, the density renders the lesion more obvious. This calcium appears to be taken from the adjacent bone, for this shows a localized but ill-defined area of decalcification of its cortex. During the second month organization of the calcium occurs, and evidence of this is seen in its frayed cotton-wool appearance. The ectopic bone is laid down in the connective tissues and not throughout the musculation in these conditions. If the affected limb is immobilized the calcium will ultimately be organized into ectopic bone and the adjacent cortex will be recalcified and receive accretions which connect the boss of new bone by an isthmus of varying breadth. For a year or more this ectopic bone will be of greater density than the bone to which it is applied, particularly if it impairs the function of the latter; but gradually it will be absorbed, only a localized thickening of the cortex remaining to indicate the site. Localized *myositis ossificans* is seen in a very small percentage of patients who have suffered contusions, even in the areas in which this lesion is most common. The theory that this ectopic bone formation is associated with a lesion of the central nervous system, sometimes but a transient lesion associated with hæmorrhage produced by the trauma, is sup-

ported by the radiographic appearances in a number of cases in which lesions of the central nervous system are known to be present.

Pyelo-Renal Backflow.

E. ROHAN WILLIAMS (*British Journal of Radiology*, September, 1941) discusses the various types of pyelo-renal backflow which may occur during retrograde pyelography. Pyelo-venous backflow is recognized in its earliest stages by a streak of opaque medium, two to three millimetres wide, passing out from the calycine angle, usually at right angles to the long axis of the minor calyx. After a short, varying, straight course this venous streak curves round towards the cortex of the kidney in the interlobar veins. Sometimes the opaque medium may be seen in the larger venous channels in the hilum, but owing to the venous flow beautiful demonstrations of the venous arcades and their interlobar tributaries are not observed as in the radiographic studies on excised specimens. Pyelotubular backflow is recognized by a fanwise streaking of fine linear radiations of the opaque medium from the papillary region of a calyx. It appears to be a mere chance occurrence and occurs independently of any rough technical procedure. Pyelo-interstitial backflow (extravasation) may occur in association with venous backflow as an ill-defined smearing of the opaque medium into the juxta-calycine region, or after forced injection as a blotchy, formless opacity, extending outwards from a calyx, or as a straight streak into the medullary tissue. The extravasation may even reach the subcapsular zone. This condition occurs only as a result of rough technique; slight degrees may occur from minimal over-distension into the connective tissues about the *sinus renalis*. Pyelolymphatic backflow is only rarely observed, but is of special interest in view of its association with chyluria of both parasitic and non-parasitic ætiology. Pyelolymphatic backflow is recognized by the visualization of one or more channels coursing from the region of a minor calyx across the pelvis in the direction of the spine over the psoas muscle.

Linear Shadows in the Lung.

FELIX FLEISCHNER, AUBREY O. HAMPTON AND BENJAMIN CASTLEMAN (*American Journal of Roentgenology*, November, 1941) present a detailed study, including differential diagnosis and pathological confirmation, of the linear X-ray shadows caused by interlobar pleurisy, plate-like atelectasis and healed infarction. Interlobar pleurisy and plate-like atelectasis are much more common than healed infarcts. Interlobar pleurisy can usually be readily differentiated from healed infarcts or plate-like atelectasis if the anatomical location of the linear shadow corresponds to the position of the interlobar fissure. It may present as a long line of equal thickness corresponding to the length of the fissure, but it is usually associated with other evidence of pleurisy, such as fluid or pleural thickening near it. Although infarcts frequently are adjacent to the interlobar pleura, they are seen as a more nodular unilaterally placed shadow near the thin line of the readily recognized interlobar fissure. Plate-like atelectasis does not occur in the location of interlobar fissures. If the linear shadow is perpendicular to one of the interlobar fissures, interlobar pleurisy is readily ruled out and the differential diagnosis lies between healed infarct and plate-shaped atelectasis. Healed infarcts and plate-like atelectasis, although often difficult to differentiate, may show the following features: the shadows of infarcts are usually shorter than atelectasis; are in a single plane, each infarct producing one line; may end in nodular extremities; and may run in any direction. The plate-like foci of atelectasis are usually long; traverse the entire portion of the lung involved, extending from one pleural surface to another; are often multiple; occur in different planes, one focus producing multiple, closely placed, nearly parallel lines; and show no nodular irregularities. In atelectasis the pleura may show a V-shaped retraction, buf there is usually no regional thickening. Other important differences between these two lesions are: (i) The linear shadow of plate-like atelectasis usually disappears within a few days, whereas that of a healed infarct persists; (ii) the diaphragmatic excursion is nearly always limited in plate-like atelectasis, but this feature is rare in a healed infarct; (iii) although both atelectasis and infarction occur predominantly in the lower portions of the lungs, infarcts, unlike atelectasis, are not uncommon in the upper lung fields.

Radiological Diagnosis of Primary Carcinoma of the Liver.

RICHARD SCHATZKI (*American Journal of Roentgenology*, October, 1941) presents a radiological syndrome which is thought to be highly suggestive of primary carcinoma of the liver. It is based on the fact that in the large majority of cases this tumour is associated with cirrhosis of the liver. The syndrome consists in the demonstration of (*a*) cirrhosis of the liver (œsophageal varices and/or large spleen with small liver); (*b*) a mass in the region of the liver; (*c*) positive evidence of cancer (metastasis). A combination of (*a*) and (*b*) alone is suggestive evidence of primary carcinoma of the liver; a combination of (*a*) and (*c*) alone is much less conclusive. A purely radiological diagnosis is not possible in those cases in which cirrhosis of the liver is not present or cannot be demonstrated by X-ray examination. A localized bulge in the diaphragm, a not uncommon normal finding, should be considered more seriously if it occurs in the presence of cirrhosis of the liver.

Retroperitoneal Pneumography.

N. W. ROOME (*The Canadian Medical Association Journal*, July, 1941) suggests retroperitoneal pneumography to aid visualization of the contours of structures lying behind the peritoneal cavity, especially the adrenals. This was carried out in 17 cases by the author according to the technique described in *The Journal of Urology* in 1935. No serious complications of any kind occurred, and valuable information was obtained about the kidneys and adrenals.

PHYSICAL THERAPY.

Neuroblastoma Sympatheticum.

G. M. WYATT AND S. FARBER (*American Journal of Roentgenology*, October, 1941) describe the radiological manifestations of neuroblastoma as observed in forty examples of this condition, verified histologically at the Boston Children's Hospital during the ten-year period ending September 1, 1939, and give certain conclusions concerning the rate of irradiation in the treatment of this disease. The ages of the patients ranged from four days to eight years and eleven months, although the majority were under the age of four years. Most of the tumours which were demonstrated radiologically were located in the posterior portion of the abdominal cavity. The posterior mediastinum was the next most frequent site of the primary tumour, and the remainder arose from the sympathetic chain anywhere from the cervical region to the pelvis. It is pointed out, however, that a definite diagnosis of primary neuroblastoma can be made only on histological study of tumour tissue. Metastases may occur to soft tissues or to bones. Most of the metastases to soft tissues occurred in the orbits or in the meninges. The metastases to bone were found in every part of the skeletal system with the exception of the metacarpal and metatarsal bones and phalanges. Any evaluation of irradiation in the treatment of neuroblastoma must take into consideration the usual survival period. Blacklock found that the average length of life of patients with adrenal neuroblastoma from the time of onset of clinical symptoms was between eight and nine weeks. Death occurred in every case. Ten of the patients in this series received radiation therapy. Five of these are still living and without evidence of disease, two to five years after treatment, even though known local metastases were present in two instances at the time therapy was instituted. Caution is indicated in the treatment of these patients, as grave reactions may follow rapid shrinkage of large tumours. It is recommended that an initial test dose of 75 to 100 *r* be given, and then subsequent doses be increased, with the severity of the reaction as a guide. Frequent blood counts should be made to determine the presence of radiation leucopenia. The total doses given were not large. It is concluded that radiation therapy should be instituted in every case after the diagnosis has been made. The prognosis, even after local metastases have occurred, is not hopeless. Radiation treatment should be tried even after metastases to bone are present.

The Influence of Röntgen Irradiation of Normal Lung on the Prevention of Subsequent Metastatic Tumour Growth.

R. P. BARDEN (*Radiology*, November, 1941) gives a preliminary report on a study designed to discover whether the susceptibility of an organ to metastases from a given tumour could be altered by Röntgen irradiation. The tumour used was the Brown-Pearce rabbit carcinoma, which grows rapidly, metastizes widely and is easily transplanted. All except one of the experiments were performed with male rabbits, since the tumour is more easily transplanted via the testis. Groups of rabbits received Röntgen irradiation to the right lung and were subsequently inoculated with the tumour. The irradiation was delivered at about 120 kilovolts and the dose varied from 300 to 900 *r*. Some groups of animals received a single dose. In others the dose was divided and given at daily or two-day intervals. Thirty-four rabbits were used in the investigation.

Six of these received tumour inoculation and no irradiation, and served as controls. Two others died immediately after inoculation from irrelevant causes. Twenty-six animals were irradiated and subsequently inoculated with living tumour. Of the rabbits not irradiated, 80% showed microscopic pulmonary metastases. In the irradiated group such metastases occurred in 35% of animals. Although the quantity of radiation varied in different groups of rabbits, all the groups showed a similar reduction in the incidence of pulmonary metastases. When metastases did occur, however, they were equally distributed in the right and left lung. Regardless of the quantity, the time at which radiation was given appeared to have some relation to the appearance of pulmonary metastases. It appeared to be more effective if given two to four weeks before than if given less than a week before inoculation. A small proportion of rabbits developed permanent immunity after removal or regression of the primary tumour. No definite cellular reaction was found round the metastatic tumour nodules in the lungs, whether radiation had been given or not. The unique freedom of the spleen in the presence of widespread metastases was a consistent finding.

Röntgen Irradiation as an Analgesic Agent.

J. R. Carty and L. M. Bond (*American Journal of Roentgenology*, October, 1941) point out that the use of Röntgen irradiation as a purely analgesic agent has not perhaps been given the attention which it deserves. Attention has been rather confined to the correction of the underlying pathological condition which causes the pain. However, Röntgen irradiation at times may be considered as an analgesic agent without thought or hope perhaps of influencing the underlying pathological condition. Pain may be caused by: (*a*) inflammation within the nervous structures themselves; (*b*) inflammation of structures in immediate vicinity of nerves or nerve endings; (*c*) pressure by outside structures, including scar tissue, upon the nerves themselves; (*d*) infiltration of the nerve structures by tumour cells; (*e*) irritation by trauma. The action of Röntgen radiation on inflammatory conditions, in reducing the size of tumours, with consequent decreased pressure, and the action upon blood vessels serving nerves are sufficient to account in a large measure for the undoubted analgesic properties of these rays. For obvious reasons Röntgen irradiation should not be used where pain is caused by fibrosis with resulting pressure on nervous structures. The authors review 240 patients suffering from non-malignant conditions and 37 suffering from advanced cancer who were treated exclusively for pain. The inflammatory conditions included superficial infections, as boil or carbuncle, *herpes zoster*, radiculitis, sciatica, myositis, arthritis, periarthritis, bursitis. Their results have been very satisfactory in *herpes zoster*. It is essential to apply treatment over the ganglia, and the results are better with the younger patients, and the earlier the treatment is given after the appearance of the vesicles, the better the result will be. There may be an exacerbation of the pain following the first few treatments, but this is a favourable sign. In sciatica and neuritis elsewhere it is important to treat both the ganglia and the nerve trunk. In malignant conditions there is sometimes a tendency to withhold Röntgen irradiation if there does not seem much hope of producing an effect on the tumour. In selected cases moderate doses may ease the pain. It is pointed out that Röntgen irradiation should be used in this way with discretion and only after much experience.

X-Ray Therapy in Tuberculous Cervical Lymphadenitis.

H. Hauser (*Radiology*, September, 1941) presents statistics of an additional group of patients to support the contention that X-ray therapy can be regarded as one of the best single agents in conjunction with well-guided surgery, which is the most practical remedy in the management of tuberculous cervical lymphadenitis. Since tuberculous lymphadenitis is an inflammatory process usually of considerable chronicity, the treatment consisted of relatively small doses of X rays given by multiple exposures at relatively long intervals and continued for a considerable time. The physical factors shown were as follows: voltage, 200 kilovolts; filtration, 0·75 millimetre of copper *plus* 2·0 millimetres of aluminium. The fractional dose of 150 *r* was given every ten days for a period of 100 days, or a total of 1,500 *r* was given. When the lesions are bilateral, 100 *r* are applied to each side of the neck at the same treatment, and the total dose is carried to 1,000 *r* to each side in a period of 100 days. In the present series the response to irradiation was so rapid in some instances that the lesion was hardly palpable after the second or third exposure. In the majority of cases the response was slower. Regardless of the rate of response, the plan was to administer a total of ten treatments. The study comprises 108 patients treated from 1932 to 1940 inclusive. All types of tuberculous cervical lymphadenitis were treated. The results of treatment are arbitrarily classified as satisfactory and unsatisfactory. Patients with satisfactory results are defined as those whose lesions underwent approximately 50% to 100% regression, whether during the course of treatment or within a few months following the last exposure, and were relieved of symptoms if any were present or of a draining sinus, and whose deformity disappeared or was considered negligible. Of the 87 patients with adequate follow-up observation, 77 were classified as showing satisfactory results and 10 were considered to be unsuccessfully treated. It is emphasized that the time-intensity factor used precludes any possibility of skin damage, even if the series of treatments are to be repeated six months or one year later.

Pain in Carcinoma of the Face, Jaws and Neck.

Donald Munro (*The New England Journal of Medicine*, June 19, 1941) has studied 30 cases of carcinoma of the face, jaws and neck with regard to the pain usually associated therewith. Contrary to the usual belief, neurosurgical procedures did not relieve the pain in the majority of the cases, only two patients being at all relieved. Pain in the cancer-bearing areas appeared to be associated with X-ray therapy. It is strongly recommended that denervation of the carcinomatous area be performed as the first step in the treatment, not only as a prophylaxis against future pain, but also as an aid to greater efficiency in the therapy of the carcinoma, because of the associated anæsthesia thus produced.

PATHOLOGY.

Cisternal Herniations of the Mid-Line of the Brain.

Ramón Carrillo (*Revista Médica Latino-Americana*, June, 1941), in an illustrated article, gives the name "cisternal hernias of the mid-line of the brain" to projections of neoplastic, tumoral, ventricular or cystic tissue into the interpeduncular cistern, cistern of Galen and pre-chiasmal cistern—this is the terminology proposed by the author for the *cisterna intercruralis superficialis, cisterna intercruralis profunda* and *cisterna chiasmatica* respectively (Rey and Retzius: *Studien in der Anatomie der Nervensystems und des Bindegewebes*, Volume I, Stockholm, 1875). In the interpeduncular cistern several anatomical forms of hernia are differentiated: (i) herniation of the protuberance, (ii) herniation of the third ventricle, (iii) herniation of the mammillary tubercle, (iv) herniation of the uncus of the hippocampus, and (v) herniation of the *gyrus dentatus*. This type of hernia does not give rise to a recognized specific syndrome, but it can produce ocular paralyses and other symptoms. The most important herniations in the mid-line are those of the cistern of Galen (*cisterna intercruralis profunda*). These are divided into four types: (i) herniation of the vermis, (ii) herniation of the cerebellar ventricle, (iii) herniation of tumours of the cerebellum, and (iv) herniation of the retrosplenial convolutions. **Clinically,** the syndrome of this type of lesion shows: (*a*) extensive papilœdema, (*b*) conjunctival congestion, (*c*) paresis of the pathetic nerve (*sindrome de pseudo-Parinaud*). Herniation into the prechiasmal cistern (*cisterna chiasmatica*) can be divided into main groups: (*a*) those which project from above downwards, where the olfactory convolutions are enclosed in the prechiasmal foramen, and (*b*) those which project from below upwards. The latter group is further subdivided into: (i) intrasellar tumours which emerge between the optic nerves, and (ii) tumours of the tubercle of the *sella turcica* which invade the cavity of the pre-chiasmal cistern; these herniations may produce alterations in the visual field by compressing the anterior border of the optic chiasma and the internal edges of the optic nerves.

Strictures of the Rectum.

Juan Antonio Garat and José Antonio Urquizo (*Revista Médica Latino-Americana*, May, 1941) divide rectal strictures into two main groups—extra-intestinal and intestinal. Those of extraintestinal origin include prostatic carcinoma, endometrioma, abscess of the pouch of Douglas, inflammatory tumours of the uterine adnexa, cysts and abscesses of the broad ligament *et cetera*. Strictures of intestinal origin may be either malignant or simple. Malignant strictures include those due to adenocarcinoma, sarcoma and lymphosarcoma. Simple strictures are grouped under four main headings: (*a*) congenital, (*b*) traumatic (including surgical, obstetrical, accidental, radium, heat, caustics), (*c*) spasmodic, and (*d*) inflammatory. The last group—inflammatory—is again subdivided into

catarrhal, specific and non-specific types. The catarrhal type is caused by *Bacillus coli*, streptococci and staphylococci; the specific type may be due to the virus of *lymphopathia venereum*, gonococcus, *Treponema pallidum*, *Mycobacterium tuberculosis*, *Entamœba histolytica*, *Bacillus dysenteriæ* (various species), diplostreptococcus and possibly Bargen's diplostreptococcus; the non-specific type may occur in diverticulitis, Crohn's disease and *"colitis tromboulcerativa grave"*.

The Action of Formaldehyde on Nervous Tissue.

FLAMINIO VIDAL AND EUGENIO E. VONESCH (*Revista Médica Latino-Americana*, July, 1941) show that, contrary to popular belief, formaldehyde has a disintegrating action on nervous tissue, especially on the phospholipoids. Using brains of man, cats and cattle fixed in formaldehyde, the authors determined the total phosphorus content (organic and inorganic) in solution. In these estimations the colorimetric method of Tisdall was used, the readings being recorded photometrically. The disintegrating action of formaldehyde is very rapid; phosphorus is found in solution within thirty minutes. Of the total dissolved phosphate, 40% to 75% is found within the first twenty-four hours; thereafter the dissolving action of the formaldehyde is constant and prolonged, and 8% of the solid matter of the nervous tissues is finally extracted. From such a solution the authors were able to recover a quarter of the total phosphorus content of the nervous tissue in question. The greatest extraction of phosphorus occurred in brains perfused with formaldehyde and in those from which the leptomeninges had been removed prior to exposure to the solution.

UROLOGY.

Prognosis in Bilateral Renal Tuberculosis.

W. F. BRAASCH AND E. B. SUTTON (*The Journal of Urology*, October, 1941) have followed up 167 patients suffering from bilateral renal tuberculosis. The survival rates are as follows: 3 years, 72%; 5 years, 58%; 10 years, 26%; 15 years, 16%. Therapeutic measures, such as sanatorium treatment, rest, diet and sunshine aid longevity. A diminution is found in the incidence of renal tuberculosis of recent years; among males the incidence of unilateral and bilateral lesions is double that among females. The discovery of the specific bacillus in the renal urine is insufficient to establish the diagnosis. There should be at least three to ten pus cells per high-power field. Pre-existent tuberculosis in other tissues often increases the resistance of the patient and may be accompanied by a relatively good prognosis.

Ureteric Obstruction in Hypertension.

J. W. PENNINGTON (*The Journal of Urology*, September, 1941) states that although much attention has been paid lately to unilateral renal sclerosis as a cause of hypertension, the question of ureteric obstruction by stricture or other cause has hardly been raised. The production of renal ischæmia by ligation of one or both ureters has been reported by Harrison and others. Such reports suggest the possibility that stasis in the renal pelvis from ureteric obstruction may be an important factor in hypertension. Wightman quotes studies of kidneys from 100 consecutive autopsies, in which pathological changes were found in the kidney in all cases in which there were more than two cubic centimetres of residual urine in the kidney. The author therefore felt that a simple method of treatment was being overlooked in certain cases of hypertension, and commenced to look for evidence of stasis in the kidney in such cases. Where such evidence was forthcoming, repeated ureteral dilatations were applied, and in an encouraging proportion of cases reduction in blood pressure was noted. The dilatation is up to 12 or even 14 F., and in some cases need only be repeated at long intervals to maintain the drop in pressure.

Control of Renal Tuberculosis.

G. J. THOMAS, T. L. STEBBINS AND S. T. SANDELL (*The Journal of Urology*, October, 1941) state that in the earliest stages renal tuberculosis is usually bilateral, but that only in more advanced cases are clinical signs manifest. In some bilateral cases surgical procedures (other than palliative) cannot be undertaken, but the patient can be rendered free of symptoms by intensive sanatorium methods. Hygienic treatment is advised only: (*a*) for patients who fail to manifest their ability to control other tuberculous lesions; (*b*) for those who have other lesions which may become life-taking; (*c*) for any who have a small unprogressive lesion; (*d*) for patients whose clinical data suggest bilateral renal involvement; (*e*) whenever the accuracy of the diagnosis is in doubt. The histories of four cases in which apparent cure has been obtained are given in detail. The authors express the belief that as renal tuberculosis is pathologically identical with lesions of other organs, the control of the destructive process should not be impossible.

E. W. BEACH AND W. G. SCHULTZ (*ibidem*) describe eight cases in which tubercle bacilli were demonstrated in the urine. Clinically these cases were not advanced, the patients having been referred on account of persistent pyuria. Clinical cure is claimed on the ground that no subjective symptoms have developed, nor have any objective signs been discovered in observation periods ranging from six to fifteen years.

Management of Cryptorchidism.

A CRYPTORCHID is defined by V. S. Counsellor (*The Journal of Urology*, October, 1941) as a testis which never descends below the external ring. Many such testes descend spontaneously at puberty; therefore operation is delayed until this time, except when the scrotum is rudimentary. "It is illogical to expect a testis to descend into a cavity which does not exist." However, the longer a patient delays after puberty, the less chance is there for successful placement in the scrotum; this is due partly to atrophy and partly to contraction of fibrous tissue behind the cord. The type of operation is determined by the age of the patient and the position and condition of the testis. Before the third decade the cord can usually be lengthened sufficiently and the testis is not generally atrophied. Therefore fixation in the pendulous portion of the scrotum is preferable by the Mayer-Thorek method if the scrotum requires enlargement, or the Cabot technique when the scrotum is normal. When the cord cannot be lengthened sufficiently to permit replacement without tension or when the testis is atrophied—as frequently happens after the third decade—orchidectomy is preferred. Hormonal therapy is reserved for comparatively few patients, and then only if spontaneous descent seems improbable. It may be beneficial in cases of hermaphroditism and hypogonadism.

Aniline (Triphenylmethane) Dyes in Interstitial Cystitis.

EDWIN DAVIS (*The Journal of Urology*, November, 1941) publishes a preliminary report on the use of aniline dyes of the triamino-triphenyl-methane group as local vesical antiseptics. This group includes those dyes known as gentian violet, methyl violet, crystal violet *et cetera*; but there is confusion because of loose terminology and multiplicity of synonyms and variation in products marketed by various firms. Many of the dyes obtainable are mixtures, rather than pure chemical compounds. The present report is an attempt to secure a simple non-operative method to alleviate the sufferings of patients afflicted by circumscribed interstitial cystitis, commonly called "Hunner's ulcer", and the dyes are used in rather concentrated form. For this reason varying degrees of immediate reaction, from direct irritation of the mucosa, are to be expected. After the reaction has spent itself relief may be expected, for quite a number of months in most cases, from the typical symptoms of pain on distension and frequency of urination. The treatment may have to be repeated at intervals. The reaction period of bladder distress may last up to ten days, but the relief which follows is unmistakable. Of 16 patients, one was made worse, but 15 benefited by varying periods of relief from three to ten months. Mostly the technique was gentle irrigation with a 1 in 20,000 solution of methyl violet; but in a few cases the opposite method of instillation with a small amount (half an ounce) of a concentrated (1%) solution, retained for five minutes by catheter spigoting, was employed. The results are equally efficacious.

Intravenous Use of Magnesium Sulphate in Uræmia.

J. C. NEGLEY (*The Journal of Urology*, September, 1941) reports on fifteen years' experience of the intravenous administration of magnesium sulphate in cases of renal deficiency when operation had just been performed or was about to be performed. At the beginning of the experiments 20 cubic centimetres of a 10% solution were injected every twelve hours for six doses, followed by a rest of twenty-four hours, on the supposition that all the drug was eliminated in this twenty-four hour rest period. The course was repeated two or even three times if necessary. Later the course was made more powerful, injections being given every six hours for three days, and so on. In blood chemistry investigations to determine renal deficiency, the blood creatinine concentration was considered more valuable than that of the non-protein nitrogen. Treatment was given when the blood creatinine was above 2·5 milligrammes per 100 cubic centimetres, but not until the non-protein nitrogen was above 60 milligrammes per 100 cubic centimetres.

National Emergency Measures.

THE TREATMENT OF BURNS.

THE following notes on the local treatment of burns were issued in England about twelve months ago. They were issued by the Ministry of Health and are based on the recommendations of the Coordinating Committee on Burns appointed by the War Wounds Committee of the Medical Research Council. They were sent to this journal by the Director-General of Medical Services some months ago. In view of some inquiries received and the present state of emergency they are published *verbatim* for the information of medical practitioners.

THE LOCAL TREATMENT OF BURNS.

1. At the First Aid Posts.

(a) Minor burns which do not require admission to hospital should, after cleansing the surface with plenty of soap and water, be dressed in the following way:

i. In the case of the face or hands, with gauze or lint impregnated with sterilized vaseline.[1]

ii. In other parts of the body with tannic acid jelly or gentian violet jelly, 1 per cent. If jellies are used no dressing should be applied over the burnt areas, and clothing must not be allowed to come into contact with the burn area until the coagulum has formed and is dry. The application of tannic acid or other coagulant jelly to the surface of a burn which has not been adequately cleansed is inadvisable.

(b) Serious burns requiring admission to hospital should be treated by the application of sterile vaseline on gauze or lint to cover the exposed portions of the burn. Provided the patient is shortly to be transferred to a nearby hospital, no attempt should be made to remove clothing in order to expose the entire burn area. Serious burns always require injections of morphine for pain, in full doses (¼-½ grain for adults).

2. At the First Hospital.

At the first hospital where the case can be fully treated the following lines of local treatment are advised for extensive burns of whatever degree.

Under gas and oxygen or "Pentothal" anæsthesia the burn areas should be thoroughly cleansed by the gentle but liberal use of soap and water, followed by saline and by the removal of débris under strict aseptic precautions (soft soap should not be used; the best application is some form of soap flakes). No violent scrubbing or scraping should be used, nor should antiseptic solutions or spirit be employed. If the burn area is impregnated with oil or grease, ether soap is of value for cleansing. Extensive burns should never be fully exposed at one time, owing to the risk of increasing shock by loss of body heat; a limited area only should be exposed for treatment, which should be completed before the next area is dealt with. Loss of heat is especially marked when ether soap is used. After the cleansing process has been completed, the burn surface and the skin around should be dried and the burn surface should be lightly dusted with sulphanilamide powder; a coagulant is then applied, except in the areas mentioned below (paragraph 4).

3. Coagulant Applications.

The following solutions are the most suitable, as they act quickly and require no special apparatus, etc.

1. Silver nitrate, 10 per cent.
2. Tannic acid, 10 per cent.
3. Silver nitrate, 10 per cent, and tannic acid 5 per cent, used alternatively.
4. "Triple dye" (one formula in common use is gentian violet, 1 in 400; brilliant green, 1 in 400; with neutral acriflavine, 1 in 1,000. The solutions are mixed in equal proportions).

The application is best made by the use of a large gauze mop; no dressings are required over the coagulated area, but it is advisable to cover the part with a sterile towel after the coagulum is dry. The edges of the area and any cracks that may develop on the surface should be dusted daily with sulphanilamide powder.

In whatever way coagulation is effected, the coagulum must cover the whole area and a good margin of unaffected skin around, and must be firm and dry or else the effect of the treatment is lost, as the patient will still lose fluid from the burn area.

4. Areas in which no Coagulant[2] of Any Kind Should be Used.

Areas in which no coagulant of any kind should be used are: the face, the hands and wrists, the feet.

Note: Circumferential tanning of a limb should be avoided if possible, and if carried out the peripheral pulse must be carefully watched for the first forty-eight hours.

Coagulants tend to contract on drying, and there is a risk of dangerous constriction of vessels or distortion of tissues, *e.g.*, the fingers and eyelids, as this occurs.

Treatment of Burns in Areas which Should Not be Tanned.

After cleansing the burn area in the manner described above, the surface is dried and dusted with sulphanilamide powder. A tulle gras dressing[3] is then applied, and this in turn is covered by six to eight layers of gauze wrung out in warm normal saline. The gauze must be kept moist by warm normal saline being dripped on it from time to time, and on no account must it be allowed to become dry. The tulle gras will enable the gauze dressings to be removed without causing pain or injury to the burn surface, provided they are kept moist; as a rule it is advisable for the first few days to change the gauze every three to four hours, the tulle gras being left undisturbed during the process. Twice a day at first, and subsequently, according to the progress of the case, once a day, the tulle gras dressing is changed; where it is possible to immerse the affected part in a bath, the tulle gras dressing is allowed to float off, which it does readily. In the case of the face or other parts for which a bath cannot be used, a gentle stream of saline greatly assists in the removal, which should be painless and not associated with bleeding. Where baths are used the limb can remain immersed for half an hour or more, the saline being kept warm throughout. Before applying a fresh dressing the unburned surface of the limb is dried and the burn area dusted with sulphanilamide powder.

Dressings must be carried out with full aseptic technique, and special care must be taken to ensure the surgical cleanliness of the arm and leg baths.

An oil silk or jaconet bag or cover is of value in keeping the gauze moist in the case of the arms and legs, both while the patient is in bed and during transport.

Where burns of the face are treated by saline packs, a jaconet bib assists in keeping the patient and the bed dry.[4]

All cases of severe burns likely to be followed by loss of function or disfigurement, or to require extensive grafting, should be transferred to a burn centre as soon as they are fit to travel.

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[1] To prepare sterilized vaseline dressings, half fill a biscuit tin with vaseline, stand the tin in a deep dish of water which is brought to boiling point on a stove or fire. When the vaseline is melted, drop into it pieces of gauze or lint about six inches square, folded in two, until the tin is full. Add more vaseline if necessary to cover the dressings. Leave the tin in the boiling water for half an hour. Then remove, put on the lid and seal with a strip of strapping.

[2] In addition to the solutions given above, tannic acid jelly and gentian violet jelly are coagulants.

[3] Tulle gras, originally a French preparation, may be prepared in the following way: Curtain net, with a mesh of 2 mm., is cut into pieces 9 cm. square. These are placed in a tin box of slightly larger size. The box is then filled with the following mixture: Soft paraffin, 96 gms.; balsam of Peru, 2 gms.; sufficient to impregnate and cover the material completely after sterilization by heat in an autoclave. The French preparation contained also halibut oil, but the value of this when applied locally is doubtful, and it can be omitted.

[4] The jaconet bib is made as follows: Take a piece of stout wire and bend it into an oval about 14 inches in its long diameter and about 8 inches in its short diameter. A piece of jaconet is then cut, of sufficient size to form a deep basin when its edges are sewn to the wire hoop. Towards one end of the jaconet basin cut a hole large enough to go over the patient's head; the edges of this hole should be turned over and sewn so that a tape can be threaded through the channel so formed. On tightening the tape the opening can be made to fit round the neck. At the other end of the basin a small hole is made into which a tube for drainage can be tied.

Medical Societies.

THE PUBLIC MEDICAL OFFICERS' ASSOCIATION OF NEW SOUTH WALES.

THE sixteenth annual meeting of the Public Medical Officers' Association of New South Wales was held at British Medical Association House, 135, Macquarie Street, Sydney, on January 22, 1942. Dr. J. Cooper Booth occupied the chair and 19 members were present.

Financial Statement.

Dr. C. E. Percy, the Honorary Treasurer, presented his financial statement for the year 1941, which was adopted. It showed receipt of £28 14s., being £22 6s. from subscriptions and entrance fees and £6 8s. interest on deposits. Expenditure was £4 14s. 6d. At the end of the year the funds totalled £569 14s. 5d., of which £400 was invested in war loan at 2½% interest, £100 in the Citizens' National Emergency Loan interest free, and £69 14s. 5d. was in bank balance and petty cash.

Annual Report.

The annual report was presented by the Secretary, and after discussion was adopted. The report is as follows.

During 1941 the war overshadowed our lives. Our Association therefore, like similar organizations, showed little activity.

There are 117 members, of whom 13, having retired from the services, are honorary members. As a result of a resolution of the last annual general meeting 35 foundation members became life members.

The committee met eight times during the year, the attendances being as follow: Dr. J. Cooper Booth (President), 8; Dr. C. E. Percy, 7; Dr. J. McManamey, 5; Dr. Dorothy McClemens, 5; Dr. S. Evan Jones, 8; Dr. Edgar Wallace, 7; Dr. G. C. Smith, 7; Dr. G. L. C. Saunders, 5; Dr. H. H. Nowland, 4; and Dr. H. H. Willis (Secretary), 8.

It is regretted that attention has again to be drawn to the appointment of a practitioner from without the services to a senior position in the New South Wales Health Department. The damaging effect of such actions upon the prestige of the services and upon recruitment is still insufficiently realized by the responsible authorities.

A change of government in New South Wales brought the Honourable C. A. Kelly to the position of Minister for Health. The Association paid its respects to him on his appointment and it is hoped our relations will be cordial.

It has been decided not to register the Association as a trades union at present. It is expected, however, that the Government will accept our membership as equivalent to membership of a union.

An attitude of general opposition has been adopted towards a plan to employ refugees in the services. It is thought that aliens, if employed at all, should be in positions an Australian is unwilling to fill and then in a temporary capacity only.

In obedience to instructions from the annual general meeting, grievances concerning working conditions and leave have been again discussed with the heads of services and it is expected that on this occasion some redress will be secured.

The Public Service Board of New South Wales had agreed to meet delegates of the Association on the occasion of the biennial salaries review, but has not yet fixed a date for the interview.

During the year a reorganization of the control of the mental hospitals in New South Wales was effected without warning and without consultation with those mainly affected thereby. On the merits of the new arrangements there is room for a diversity of opinion, but the methods adopted are much to be deplored. It is felt that the present arrangement is transitional and it would be well for members to determine what permanent system of control would be in the best interests of their patients and themselves.

The committee has not expressed any opinion on the schemes for a national medical service put before the profession by the National Health and Medical Research Council and by the Federal Council of the British Medical Association and looks to members for guidance on this important matter.

Leave and Conditions of Service.

Dr. Clifford Henry, pursuant to notice, moved a long resolution, which was carried, concerning leave and conditions of service. In doing so, he apologized for mentioning such a matter when the enemy was approaching our shores, but pointed out that the attitude of the authorities on this matter had been unsatisfactory for many years, and while the lot of all other workers was being improved, that of public medical officers was becoming worse each year. He noted in the annual report that the committee expected some good to come from its representations of a few months ago, but he hoped the matter would not be allowed to drop, but be even more vigorously pursued in the future.

Health Service for the Community.

A discussion on schemes for a health service for the community revealed little diversity of opinion. It was generally agreed that public medical officers had no personal interests at stake and so could take a more impartial view of the problem than other sections of the profession. The following motion was finally adopted as expressing the consensus of opinion of the meeting:

> That a salaried medical service would provide the best health service for the community and with proper safeguards, such as medical control, would also be in the best interests of the medical profession.

Election of Officers.

The election of officers for the year 1942 resulted as follows:

President: Dr. A. T. Edwards.

Honorary Secretary: Dr. H. Hastings Willis.

Honorary Auditor: Dr. S. McGeorge.

Committee (elected by groups): Dr. C. E. Percy, Dr. J. McManamey and Dr. G. C. Smith (New South Wales Health Department), Dr. S. Evan Jones and Dr. A. T. Edwards (Mental Hospitals), Dr. Dorothy McClemens (Education Department), Dr. H. M. Taylor (Works and Railways) and Dr. H. H. Willis (Repatriation Department).

Medical Practice.

INDICATIONS FOR THE INTRAVENOUS ADMINISTRATION OF FLUID.

THE following statement on the indications for the intravenous administration of fluid has been received from the Blood Transfusion Committee of the New South Wales Division of the Australian Red Cross Society.

The three principal conditions in which fluids may need to be administered intravenously are shock, burns and hæmorrhage. In all three conditions there is a common factor, namely, a diminution of the volume of the circulating blood causing failure of the peripheral circulation. Indications for the use of blood serum and saline solutions will be discussed in this memorandum.

Shock.

There are two forms of shock: (*a*) primary and (*b*) secondary, and their symptoms are similar.

Primary shock sets in immediately following an injury. It is probably due to afferent impulses causing reflex vasodilatation and a profound fall of blood pressure. The diminished blood supply to the brain may result in loss of consciousness. After a time recovery takes place or else the condition may merge into secondary shock.

Secondary shock: Various factors play their part in the induction of secondary shock. They are pain, injuries to internal organs and to the central nervous system, hæmorrhage, prolonged anæsthesia, exposure, fatigue, cold, fear and absorption from traumatized tissues. These factors, acting in varying degrees and combinations, produce a fall of blood pressure which, if continued long enough, produces a vicious cycle which is often fatal. This vicious cycle shows the following features: low blood pressure, capillary stasis and dilatation, reduced blood volume. The last mentioned is the biggest factor in producing low blood pressure and circulatory failure. It is important to emphasize the time factor; if greatly reduced blood pressure has been present for any considerable period, spontaneous recovery is often impossible. In other words, the vicious cycle is so firmly established that it may be broken only with great difficulty or not at all. Other pathological changes in secondary shock are anoxæmia, due mainly to capillary stasis, and increased capillary permeability resulting in loss of fluid into the tissues. All of these phenomena produce a progressive fall of blood volume and so perpetuate the low blood pressure. It is the profound deterioration in the circulatory system and the summation of the various effects

produced on the tissues that give rise to the peculiar picture of secondary shock.

The treatment of secondary shock depends on the restoration of the circulation as completely and as soon as possible. It is therefore necessary that a fluid be given which will be retained in the vessels and not transude into the tissues. Whole blood, plasma and serum are such fluids. The proteins of whole blood and of human serum act by exerting an osmotic effect. The effects of saline solutions on the other hand are short-lived, since the saline solution is not retained within the vessels.

Hæmorrhage.

The symptomatology of hæmorrhage is due to a sudden reduction in the volume of circulating blood followed by a fall in the hæmoglobin percentage. In an acute hæmorrhage the first is by far the more important. If a large volume of fluid is removed from the circulation, the body attempts to compensate by drawing on its reserves. This is further assisted by selective vaso-constriction, but a stage may be reached when these compensatory mechanisms fail. The loss of the red blood cells is to some extent compensated by the entry into the circulation of blood cells from reserves in the spleen, bone marrow and liver *et cetera*. However, this cannot be brought about unless the volume of the circulating blood is sufficiently maintained.

Burns.

Various factors enter into the symptomatology of burns, but two are outstanding, namely, shock and toxæmia.

Shock.—There are two stages: (a) Primary, similar to that found in traumatic or post-operative shock, but as the causative factors of secondary shock are more likely to be present, primary shock frequently passes into secondary shock. Therefore symptoms should be treated very energetically in the primary stage.

(*b*) Secondary shock. It has been shown that there is a marked loss of blood plasma from the burnt area which leads to concentration of blood and a marked decrease in the circulating blood plasma. The cardiac output falls and the peripheral circulation tends to fail.

Toxæmia.—Toxæmia occurs at a slightly later stage and is believed to be due to the absorption of toxic substances produced by autolysis of proteins in the burnt area.

Fluid for Intravenous Administration.

Blood.

The ideal way of administering blood is to use freshly collected blood. But in recent years blood has been stored, and when fresh blood is not obtainable stored blood may prove a valuable substitute in metropolitan hospitals. Its life time, however, is definitely limited, and under active service conditions the use of stored blood from blood banks has proved a dangerous procedure owing to the risk of bacterial infection. Its use is now practically banned in the Middle East, where the use of stored blood over thirty-six hours old is completely forbidden.

Once collected, stored blood should not be agitated until immediately before use. Careful observation should be made regarding hæmolysis, which is visible as a reddish zone above the buffy layer of leucocytes on the surface of the sedimented corpuscles. Any specimen of blood showing more than a very slight layer of hæmolysis should on no account be used. Stored blood must be kept at a temperature between 2° and 4° Centigrade. If time permits, blood taken from storage may be warmed by immersion in a flask of lukewarm water at a temperature not higher than 37° Centigrade (98° F.) for about twenty minutes.

In malarial countries where the A.I.F. is now engaged, transmission of malaria has been found to be such a danger that curative, not prophylactic, doses of quinine are administered with every blood transfusion. This risk does not arise in the case of pooled human serum.

It is always desirable to use donors belonging to the same group as the recipients, but where there is not possible it is permissible to use the universal group "O" donors for transfusing patients of other groups. Direct cross-matching tests between the patient's serum or plasma and the donor's red cells is a safeguard against errors in the official blood grouping.

Pooled Human Serum.

Pooled human serum has the advantage that it may be administered to any patient without blood grouping.

Serum is supplied in two forms: (a) wet and (*b*) dry.

Wet serum is supplied in "Soluvac" bottles in quantities of one litre. It is a clear amber-coloured fluid. A precipitation occurs which causes some turbidity, but is not a cause for rejection of the serum. It makes the use of a filter necessary, however, when the serum is administered. Leaking bottles should not be used. Rejected bottles should never be discarded or emptied of their contents, but returned to the base for investigation. If it is desired to warm the serum before administration the metal and rubber disks should be removed before the "Soluvac" flask is placed in the water bath. If this is not done, expansion of the contents of the flask, with the rise in temperature, will cause a quantity of the serum to flow up the glass tube and out of the flask when the two disks are removed.

Dry serum is at present prepared in amounts of 200 cubic centimetres in 12-ounce flat dispensing medicine bottles. The level of the original volume of serum is marked with an arrow on the bottle, which is sealed with a screw cap and rubber washer. A bottle containing 600 cubic centimetres of sterile specially prepared pyrogen-free distilled water is issued with each three bottles of dry serum. To reconstitute the serum the caps are removed from the water bottle and three serum bottles and placed with their under surfaces on a clean swab. The necks of the bottles are wiped with a swab moistened with methylated spirits. Distilled water is poured into each serum bottle to the level of the arrow mark, the caps are replaced and the bottles shaken until the powder is dissolved. Turbidity due to lipoids is always present in reconstituted dry serum and is not a cause for rejection. The reconstituted serum is poured back into the water bottle and the special sterile bung, fitted with a glass tube, is inserted. The metal connexion of the standard giving set supplied in the transfusion pannier is inserted into the vacant hole in the rubber bung. A blood filter must always be incorporated in the giving set. The bottle is now inverted and hung up about three feet six inches above the patient and the serum administered in the usual way. In an emergency it may be given through a sterile funnel (containing several thicknesses of gauze) attached to rubber tubing and needle. Dry serum keeps indefinitely if it is stored in a cool dark place. It should be used immediately it is reconstituted. For moderate hæmorrhage or shock the dose recommended is three bottles or 600 cubic centimetres. For severe hæmorrhage or pronounced shock the dose is at least five to six bottles or 1,000 to 1,200 cubic centimetres.

Saline Solutions.

Saline solutions are supplied in the Army pannier as normal saline, 5% dextrose in normal saline and Ringer's solutions. As a large number of flasks, especially those containing dextrose saline, have been found to be visibly contaminated with fungi under active service conditions, care should be taken that each flask is carefully examined before use and that instructions regarding the presence of a vacuum in the flask are strictly followed.

The Treatment of Hæmorrhage, Shock and Burns by Intravenous Administration.

Hæmorrhage.—Freshly collected blood is the method of choice, as this will achieve both objects of treatment, namely, restoration of blood volume and restoration of red blood cells and hæmoglobin percentage; but on active service this has often been found to be impracticable. Stored blood may also be useful, but the dangers already mentioned must be borne in mind. As previously stated, the primary object is the restoration of blood volume. Serum will achieve this very satisfactorily, but it may be necessary at a later stage to administer whole blood to overcome the secondary anæmia which may follow. When available, serum can be administered more promptly than blood and will often save lives.

Saline solutions are less satisfactory than serum, as their function in restoring the blood volume is short-lived, the fluid rapidly passing into the tissues. They are valuable, however, whilst blood or serum is being obtained. Excessive quantities of saline may result in "water-logging" of the patient, and this is a constant source of danger. If saline is administered, careful attention should be paid to the condition of the patient's lungs as the first signs of "water-logging" are detected by the finding of fine râles at the bases of the lungs.

Shock.—When shock is associated with hæmorrhage, blood is the medium of choice. In cases, however, of severe shock without hæmorrhage, serum should be used, as there has been no loss of red blood cells or of hæmoglobin. The aim is to restore the volume of the circulating blood. By doing this, stagnant red blood cells will be washed into the circulation from the periphery. Blood is less satisfactory than serum, as the introduced red cells are unnecessary and will tend to stagnate also. In an attempt to overcome the vicious circle which may be set up in shock, serum should be administered early in sufficient dosage and at a

rapid rate. The remarks in regard to the use of saline solutions in the treatment of hæmorrhage apply also in the treatment of shock.

Burns.—As there has been no loss of red blood cells and as there has been a known loss of proteins and fluids, serum is the medium of choice in the treatment of burns. As primary shock is so apt to develop into secondary shock, serum should be administered early and should not be delayed until secondary shock has actually developed. The replacement of lost protein is a further indication for use of serum, and local applications should be directed to prevention of further loss. If serum is available, blood is rarely indicated in burns, but, of course, it is a valuable substitute and should not be withheld. The administration of saline solutions may be necessary in burns to overcome dehydration. Concomitant administration with serum is sometimes necessary. Again, careful watch should be kept for "water-logging".

Rate of Administration.

It is very difficult to lay down any definite principles regarding the rate of intravenous administration of fluid, as so much depends on the clinical condition of the patient and his reaction to treatment. In general, however, it can be said that for the treatment of acute hæmorrhage and for the treatment of severe shock, serum or blood should be administered as rapidly as possible. On the other hand, patients with chronic anæmia should be given fluid at slower rates in order to prevent overtaxing the heart. In shock and acute hæmorrhage the initial dose should be large and a dose of less than one litre is rarely indicated. Reactions during or following blood transfusion are thought to be usually due to dissolving the citrate in faulty distilled water or to failure to clean thoroughly the glass and rubber parts of the equipment. At times, however, they may be due to minor incompatabilities of the blood. Small doses of adrenaline or ephredine may be given to counter the reactions and symptomatic treatment—hot-water bottles, warm blankets, morphine *et cetera*—should be given. The urine should be tested at regular intervals following the administration of blood or serum, and any hæmoglobinuria is an indication for the immediate administration of large doses of alkali, such as potassium citrate. Reactions are treated symptomatically.

The standard blood filter supplied in the pannier is necessary when giving (*a*) recently collected blood in which it is possible that clots are present, (*b*) stored blood (blood bank), (*c*) serum, and (*d*) reconstituted dried serum.

Dosage.

Reports show that casualties occurring under service conditions and during air raids often require continued administration at short intervals of serum, or serum and blood, or blood alone in order to restore the blood pressure to the normal range and so counter secondary shock. Several litres may have to be given during twelve or twenty-four hours.

In all cases in which blood, serum or saline is administered, fluid balance charts showing details of intake and output, accurate records of blood pressure estimations and, if possible, hæmoglobin values, should be kept.

THE FLYING DOCTOR SERVICE OF AUSTRALIA.

THE annual meeting of the Federal Council of Australian Aerial Medical Services was held at Melbourne on Friday, February 27, 1942. The meeting was attended by representatives of the six State sections.

Change of Name.

A special resolution was adopted changing the name of the organization to the Flying Doctor Service of Australia. This change of name will be ratified in the near future by each of the six State sections which control a "Flying Doctor Base".

Annual Report.

The annual report covering the year ended December 31, 1941, was adopted. The report is as follows.

In submitting the report for the year we are pleased to be able to report that each section—with the valued support of its contributors and others, and the able management of its council, committees and staffs—has been able to carry on the good work of the Flying Doctor Services.

In common with other organizations and individuals, the year has not been free from difficulties and problems; in some cases this has meant certain departures from usual practice. With the coming months many of the bases may be faced with even greater difficulties, but all will go forward with the hope that, come what may, the Flying Doctor Service will carry on its work in the interests of those in the outback.

Aerial Ambulance and Wireless System.

Hereunder will be found particulars of the mileage flown by the Aerial Ambulances, and the telegraph traffic (commercial telegrams) which have passed through our wireless bases for the year ended December 31, 1941.

It is recognized, of course, that, in addition, during the "medical sessions" which are held at regular intervals during each day, our doctors give medical advice, by wireless, to patients at the outposts.

	Commercial Telegrams. Messages.	Medical Flights. Flights.	Miles.
Cloncurry	17,737	84	42,294
Broken Hill ..	5,642	75	36,405
Alice Springs ..	8,541	17	7,044
Kalgoorlie	4,188	13	6,310
Port Hedland ..	5,523	78	20,447
Wyndham	10,758	33	17,558
	52,389	300	130,058

There are approximately 250 wireless transmitting and receiving sets linked with one or other of our bases. These, with the landing grounds at the outposts, have always been considered of great importance to Australia, but never more than at the present time, when Australia, particularly the north, is threatened.

When referring to the above six flying doctor bases of our organization, mention might also be made of the aerial ambulances and flying doctors at Katherine (under the Federal Government's Northern Territory Medical Service) and at Ceduna, South Australia (Bush Church Aid Society); both these services are doing splendid work in their respective spheres. It is with regret that we report the unfortunate accident at Katherine last year. Owing to difficulties in obtaining a suitable plane, this service has ceased operations temporarily.

Cloncurry Base (Queensland Section).

During the year the Queensland Section purchased a suitable house as residence for the doctor. The council, with its wireless committee, has for some months been considering the question of rebuilding the base wireless station. In all probability the base may be removed to a more suitable site; a new transmitter, to replace that which has done good service for very many years, will be installed. The council is also considering the possibility of building an operator's residence.

The establishment of the sub-base at Charleville is also under consideration, but with the present difficulties in obtaining medical assistance, aircraft *et cetera*, it may be necessary to postpone establishment of the base until a later date.

The council is to be congratulated on the publication of its first issue of *The Flying Doctor*, an interesting magazine, which they propose to publish quarterly.

Broken Hill Base (New South Wales Section).

The section's aerial ambulance, L. M. Pattinson, has been doing good work. One problem facing the section is the servicing of the plane. Notwithstanding every effort, the New South Wales Section has found it impossible to obtain a suitable mechanic; thereby additional miles must be flown and heavier expense incurred through the necessity for arranging periodical flights to Adelaide or Sydney for examination and overhaul.

In reporting on the voluntary contribution scheme established by the New South Wales Section, it is interesting to note that during the year 80 subscribers paid no less than £645 3s., fifteen of the subscribers being treated by the doctor.

The New South Wales Section recently entered into an arrangement with the State Government whereby, in payment of a small additional subsidy, the doctor at Broken Hill base gives wireless advice daily to Tibooburra District Hospital, some 200 miles away, and makes a regular flight monthly, in addition to any special flight which may be necessary.

Alice Springs Base (South Australian Section).

During the year the Alice Springs Base, which had been in operation for some eighteen months, was officially opened. The main ceremony was in Adelaide, but Dr. George Simpson was invited by the South Australian Section to visit Alice Springs and unveil the commemorative tablet at the base.

Kalgoorlie Base (Eastern Goldfields Western Australian Section).

As at other bases, the base at Kalgoorlie has carried on efficiently. It must be recognized that its constituency is rather different from those of other bases. The local council is to be congratulated on the various local efforts arranged to supplement its share of the Federal Council's income and to cover its expenditure.

Port Hedland (Western Australian Section).

During the year the Western Australian Health Department and the Western Australian Section were faced with the problem of appointment of a doctor in view of the forthcoming completion of Dr. Dicks's term of appointment. Dr. Dicks had stated his willingness to continue for a further term, on condition that he—a fully qualified aviator—be permitted to act as his own pilot for the Fox Moth Aerial Ambulance, which it was suggested should be purchased by the Western Australian Section. After giving the matter every consideration the council of the Western Australian section agreed to the proposal, although it recognized that it was preferable to have an aeroplane under charter from an aviation company if possible. The decision of the section has ensured the appointment of a doctor at Port Hedland for a further term, and at a time when the Health Department was having great difficulty in filling vacancies along the west coast. The above plane is the second to be owned by a section of Australian Aerial Medical Services, but Dr. Dicks is the first doctor in the service to act as pilot. Other sections will follow with interest the work at Port Hedland.

Wyndham Base (Victorian Section).

The absence of a permanent doctor at Wyndham has been the main problem facing the Victorian Section and also the Health Department. Early in 1941 the doctor resigned. After a brief period a young doctor from Victoria, who happened to be passing through Wyndham, was prevailed upon by the town folk to remain for the meat works' season. After his departure, the department still unable to make a permanent appointment, it was possible to arrange for the cooperation of Dr. Jean Davies, of the Presbyterian Church of Victoria, who was visiting Kunmunya Mission, one of the outposts linked with Wyndham. Her advice by wireless was very helpful on many occasions. During December a doctor considered most suitable applied for the position at Wyndham and steps are being taken to obtain his release from service with the military forces.

Federal Government.

During the year the council submitted its estimates to the Federal Government's committee. On the change of government, in reply to our letters of goodwill, letters expressing appreciation of our organization were received from the Prime Minister and certain of his colleagues.

Finance.

The Federal Council would again take this opportunity of expressing its thanks to the Federal Government for its subsidy and its appreciation of the interest shown by the trustees of the H. V. McKay Charitable Trust, who again made a grant of £1,000 to the Federal Council. It would also acknowledge the generous assistance rendered to sections by certain State Governments and contributors both great and small.

In this connexion it is interesting to note that the total cost of maintaining our six bases—excluding the salaries of the doctors at Alice Springs, Port Hedland and Wyndham, who, being government medical officers, are paid by the health departments—amount to £21,000. Of this amount, £10,800 is received from Federal and certain State Governments.

In closing this report, on behalf of the Federal Council I would express appreciation of the work of our flying doctors over the years that have passed—our thoughts are particularly with those now serving with His Majesty's forces at home and abroad. We are also grateful to our pilots and wireless operators, some of whom have been granted leave for service in the Royal Australian Air Force, while others, also anxious to enlist, have refrained from doing so on account of the nature of their present duties.

W. L. Sanderson,
President.

Naval, Military and Air Force.

APPOINTMENTS.

The undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 70, of March 5, 1942.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Appointment.—Leonard Hugh Catchlove is appointed Surgeon Lieutenant, dated 3rd February, 1942.

Australian Military Forces.

AUSTRALIAN ARMY MEDICAL CORPS.

Northern Command.

First Military District.

Lieutenant-Colonel B. F. R. Stafford relinquishes command of a Field Ambulance and is transferred to the Reserve of Officers, 10th January, 1942.

The following officers are appointed from the Reserve of Officers and to be Captains (provisionally): Honorary Captains G. H. Ellis, 30th December, 1941, C. A. C. Leggett, 6th January, 1942, V. R. Woodhill and M. G. Wilson, 26th January, 1942.

To be Captains (provisionally).—George Atkin Sampson and Ian Hamilton MacGillivray, 6th February, 1942.

The date of appointment of Captain (provisionally) K. B. Shallard, notification of which appeared in Executive Minute No. 20/1942, promulgated in *Commonwealth Gazette* No. 28 of 1942, is amended to read "5th January, 1942".

Eastern Command.

Second Military District.

Honorary Captain N. G. Malouf is appointed from the Reserve of Officers and to be Captain (provisionally), 1st January, 1942.

To be Honorary Captains.—John Antill Pockley, Geoffrey Henry Puddicombe, George Alexander Chambers, Kevin James Collins, Basil Diethelm, Sherwood Arthur Horsman, Carl John Zimmerman, Harry Mathew John Windsor, Leonard Thomas Shea and Ewen Neil McQueen, 30th January, 1942.

Southern Command.

Third Military District.

Captain (provisionally) V42516 A. B. Hewitt is transferred to the Reserve of Officers (A.A.M.C.), 23rd January, 1942.

The following officers are appointed from the Reserve of Officers (A.A.M.C.) and to be Captains (provisionally): Honorary Captains A. E. Piper, 2nd January, 1942, K. D. Anderson, D. A. Kidd, M. M. Velik, G. R. A. Syme, F. P. Donoghue, A. R. K. Kelly and A. E. Brauer, 1st February, 1942, and J. W. Wales, 2nd February, 1942.

Honorary Captain W. R. Maloney is appointed from the Reserve of Officers (A.A.M.C.), 5th Military District, and to be Captain (provisionally), 1st February, 1942.

The resignation of Honorary Captain C. H. Chambers of his commission is accepted, 1st December, 1941.

To be Honorary Captain.—Heriot Roland, 26th January, 1942.

Royal Australian Air Force.

Citizen Air Force: Medical Branch.

The following are appointed to commissions on probation with the rank of Flight Lieutenant with effect from the dates indicated: Thomas Joseph Fennell, M.B. (3864), 20th January, 1942; Leopold Rupert Flynn, M.B., Ch.M., M.R.A.C.P. (3940), 22nd January, 1942; Clive William Kingston, M.B., B.S. (3949), David Weddell Short, M.B., B.S. (3944), Edward Coles Swan, M.B., B.S. (3943), William John Patrick Woolcock, M.B., B.S. (3946), 2nd February, 1942.

The following Flight Lieutenants are transferred from the Reserve to the Active List with effect from the dates indicated: G. Buckland (2751), A. W. Bull (3401), 26th January, 1942; R. L. Douglas (2724), G. A. Hodgson (3612), 2nd February, 1942.

Reserve: Medical Branch.

The following are appointed to commissions on probation with the rank of Flight Lieutenant with effect from the dates indicated: David Arthur Brown, M.B., B.S. (3887), Ross Byrne, M.B., B.S. (3888), Roger Henry Edwards, M.B., B.S. (3889), John Hilton Newton, M.B., B.S. (3890), Francis Harold Read, M.B., B.S. (3891), 22nd January, 1942; Justin

Paul Fleming, M.B., B.S. (3942), Leslie John Ray, M.B., B.S. (3941), 26th January, 1942; Alexander James Kerr, M.B., B.S. (2906), 27th January, 1942.—(Ex. Min. No. 42—Approved 26th February, 1942.)

CASUALTIES.

ACCORDING to the casualty list received on March 10, 1942, Major D. G. Croll, A.A.M.C., of Brisbane, Queensland, is reported removed from seriously ill list.

Captain B. H. Hocking, A.A.M.C., of Hawthorn, Victoria, is reported killed as the result of enemy action.

DECORATIONS.

CAPTAIN J. L. TAYLOR, A.A.M.C., has been awarded the Military Cross. The official report of his award contains the following statement.

> This officer was Regimental Medical Officer of a unit and the courageous manner in which he moved about the battlefield under all conditions of fire both day and night and the coolness with which he performed his duties excited the admiration of all ranks. By this courage and devotion to duty he inspired all ranks with confidence and his complete disregard for his personal safety deserved recognition.

Correspondence.

"THE MISSING LINK IN DEMOCRACY."

SIR: You kindly granted me a review of the above volume in the issue of February 7, 1942. Professor Chisholm, reviewer on sociological subjects for the Melbourne Press and publishers, has favoured me with the following opinion, which stresses the viewpoint of the non-medical student of the subject.

Professor Chisholm states:

> In appreciation of your book I find it an extremely penetrating and creative investigation, and think it provides a feasible solution for some of the most troublesome problems and dilemmas of democracy, and it avoids all the fanaticisms, leaving a place for some of the urges—such as religion—which are deeply rooted in mankind, whether they are rational or not. And it is most interesting to find the biological question brought in, as this latter is so often neglected in sociological studies.
>
> The advisory panel idea, I think, would function splendidly in Australia, and is admirably constructive. It would function here because, whatever social insanity may still persist in this country, the whole tradition of the place would make it difficult for it not to function. There may be, and in fact undoubtedly are, even in Australia, some money-makers who put their own particular urge before all else; but even the worst of them do not escape the healthy contagion of the public ethos, and are ready to admit the need for a "brave new world".
>
> A splendid proof of the validity of your concept in a particular case is given by the Labour appointment of a Broken Hill Pty. man to such an important post. It does credit to both parliamentary parties, and leaves one with high hopes of the possibilities out here. Can we venture to hope that this part of your scheme (and other parts) might be tried in Australia, so that an example could be given to the world of what can be done without sacrificing democratic principles.
>
> The shortest road to economic justice is probably the Russian one; but as your book demonstrates, there are many more things than economic justice to be considered. We have to preserve liberty, for instance, and even a reasonable amount of private enterprise—so long as it is kept free from greed, and that is possible only by means of law.
>
> Possibly the solution of humanity's chief problems lies in a synthesis of the best of the Russian ideas and the best of the democratic ones. And among the democratic ones, the leading ideas put forward in your book take a very high place. That is probably the most sincere praise I can give it. I would call it a work of human socialism, as distinguished from inhuman socialism, and human socialism is the only thing in my opinion that will ever make man reasonably happy.
>
> I can assure you that your book gives me real pleasure from another point of view also. You will forgive me if I say that a good few scientific and medical men see things too exclusively through the eyes of their specialisation. But such books as Alexis Carrel's *"L'Homme, cet inconnu"* have given me new heart. We can get nowhere until science and humanism collaborate; and it is very pleasing indeed to find in your book one more harbinger of this coming synthesis. We have lost something that the Middle Ages, despite their faults, had possessed—a wholeness with regard to life. You get pretty close at times to the social synthesis that had been evolved (with all sorts of defects caused by ignorance and superstition) in the Middle Ages, with their guilds and their unified and to some extent socialised society. The Greeks had it too. And man can never get the full benefits of the Renaissance until we find it again without sacrificing the scientific and intellectual exactnesses that that great movement gave us.

In conclusion I would like to add a quotation from the preface of the volume.

> Though a main purpose is to suggest a democratic manual for a more humane and reliable system of peace-time living, the general principles entailed should provide a finger-post to a more direct and time-saving policy, in the conduct of a scientific war.

Yours, etc.,

F. L. TRINCA.

96, Balaclava Road,
Caulfield,
Victoria.
March 3, 1942.

Obituary.

ALICK MURRAY-WILL.

DR. ALICK MURRAY-WILL, whose death was reported in this journal some weeks ago, was a graduate of the University of Aberdeen, qualifying as Bachelor of Medicine and Master of Surgery in 1888. He was a distinguished student; among the other honours that he gained was a prize in pathology in his final year. In his year were men who afterwards reached high distinction. Among them were Sir Arthur Keith, Sir Leslie MacKenzie and McKerron, who became professor of midwifery in the University of Aberdeen. After graduation Alick Murray-Will became house surgeon in the Aberdeen Royal Infirmary, serving under the professor of surgery, Sir Alexander Ogston. After leaving Aberdeen he went to London for post-graduate study; he afterwards joined the Orient Steam Navigation Company, Limited, and made several voyages to Australia as medical officer. In 1892 he left the sea and commenced practice in Sydney. Three years later he was appointed to the honorary staff of the Sydney Hospital and was identified with it until his death. He was always quiet and unassuming, but his work was sound and his attention to detail unremitting. He was one of a band of physicians whose devotion, whether in outdoor or indoor service, set the medical side of Sydney Hospital's achievements on a high plane. The hospital and the public of Sydney stand to this day in Alick Murray-Will's debt.

Dr. A. W. Holmes à Court writes:

> With the passing of A. Murray-Will the profession is poorer by the loss of a highly educated and cultured gentleman whose life was one of unobtrusive usefulness in the service of the sick.
>
> Apart from an extensive practice in general medicine, the major part of his activities was devoted to honorary service at the Sydney Hospital. As out-patient assistant physician from his appointment in 1895 until he became physician to in-patients in 1919 he gave conscientious service for twenty-four years. It was unfortunate that when he reached the full staff only three years remained before his retirement. This fact, however, detracted nothing from his zeal and enthusiasm for his work, and he was well known and highly respected by generations of residents and students who were fortunate enough to be associated with him. From 1921 he remained honorary consulting physician to the hospital. Of highly sensitive temperament and retiring disposition, he did not seek publicity and was happy in enjoying the goodwill of his colleagues and the affectionate regard of all those with whom he came in contact. His

life and work were a tradition of service to the hospital, and he belonged to the elect band of cultured physicians whose reward lay in the knowledge of work well and truly done. His son Dr. Ewen Murray-Will is at present on service with the Australian Imperial Force.

GEORGE LEONARD LINDEN.

We are indebted to Dr. Colin Laing for the following appreciation of the late Dr. George Leonard Linden.

The recent death of Captain George Leonard Linden on active service in Malaya terminated the career of a young doctor that was full of promise.

George Linden was educated at Geelong Grammar School, Trinity College and the University of Melbourne. At school he was prominent in athletics and was a school prefect. During his medical course his name appeared in the honours list on several occasions, and he was successful in passing the preliminary examination for the Fellowship of the Royal College of Surgeons on the last occasion it was held here.

As resident medical officer at the Alfred Hospital in 1941, his cheerful and enthusiastic disposition as well as his zeal in carrying out his work made him an outstanding and well-liked member of the staff. At the end of 1941 he left the Alfred to take up his commission with the Australian Army Medical Corps. His death will be felt alike by his confrères and associates, who were, without exception, attracted by his good fellowship and charm of manner.

WALTER FERGUSON STRAEDE.

We regret to announce the death of Dr. Walter Ferguson Straede, which occurred on March 9, 1942, in Central Australia.

WALTER PERRY.

We regret to announce the death of Dr. Walter Perry, which occurred on March 12, 1942, at Darlinghurst, New South Wales.

MEDICAL WAR RELIEF FUND.

The following is a twenty-third list of contributions to the Medical War Relief Fund established by the Federal Council of the British Medical Association in Australia for the relief of distressed medical practitioners in Great Britain.

New South Wales.

£5 5s.: Dr. Stratford Sheldon.
£5: Dr. K. F. Potts.
£1 1s.: Dr. Reginald Power.

Medical Appointments.

The following have been appointed to act as coordinating officers for the hospital, ambulance and first aid services in the civil defence organization of Queensland in the cities, towns and shires as follows: Coolangatta, Dr. Litton Arthur Armitage Forbes; Southport, Dr. Wallace Arthur Arundel Mackey; Ipswich, Dr. Hamilton Stuart Patterson; Warwick, Dr. John Sands Smyth; Toowoomba, Dr. Glenioth Victor Hickey; Maroochy, Dr. Francis John Short; Gympie, Dr. Robert Cecil Black; Kingaroy, Dr. William John Saxton; Maryborough, Dr. Roy Stanley McGregor; Bundaberg, Dr. Duncan Fowles; Gladstone, Dr. Harrison Booth Skinner; Rockhampton, Dr. Reeve Palmerston Rundle; Mount Morgan, Dr. Eric Roderick Watkins; Sarina, Dr. John George Morris; Mackay, Dr. Philip Scott Woodruff; Proserpine, Dr. Percy Alexander Stevens; Bowen, Dr. William Edward Lindsay; Ayr, Dr. John Albert Emmett; Townsville, Dr. Leslie Halberstater; Charters Towers, Dr. Thomas Roy Edmeades; Hinchinbrook, Dr. Gordon Carey Morrissey; Cardwell, Dr. Maurice Leslie Unwin; Johnstone, Dr. Timothy John Cotter; Mulgrave, Dr. Alfred Meillon Langan; Babinda, Dr. Thomas White Carroll; Atherton, Dr. Clive Lansdell Paine; Douglas, Dr. Thomas Joseph Flynn.

Dr. David William Johnson has been appointed Coordinating Officer for the hospitals, ambulance and first aid services in the Brisbane civil defence organization, Queensland.

Dr. Neil Douglas Campbell has been appointed Government Medical Officer at Bowraville, New South Wales.

Australian Medical Board Proceedings.

QUEENSLAND.

THE undermentioned has been registered, pursuant to the provisions of *The Medical Act*, 1939, of Queensland, as a duly qualified medical practitioner:

Winterbotham, Harold John, M.B., B.S., 1941 (Univ. Melbourne), General Hospital, Brisbane.

Diary for the Month.

MAR. 24.—New South Wales Branch, B.M.A.: Council Quarterly.
MAR. 26.—New South Wales Branch, B.M.A.: Annual Meeting.
MAR. 26.—South Australian Branch, B.M.A.: Branch.
MAR. 27.—Queensland Branch, B.M.A.: Council.
MAR. 27.—Tasmanian Branch, B.M.A.: Council.
MAR. 31.—New South Wales Branch, B.M.A.: Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

Editorial Notices.

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All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility unless such a notification is received within one month.

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